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AETIOLOGICAL INCIDENCE OF HEART DISEASE IN BOMBAY—ANALYSIS OF 4335 AUTOPSIES

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During the period beginning 1st January 1926 and ending 31st December 1939 a series of 4,335 consecutive autopsies were performed in the Department of Pathology and Bacteriology of the K. E. M. Hospital. These autopsies were done either by the Resident Pathologist or the Assistant Clinical Pathologist of the hospital under the direction of the Professor of Pathology. An analysis of the data presented by the autopsy protocols was undertaken with a view to get some idea regarding the aetiological incidence of heart disease. Cases in which atheroma of the aorta was the only cardiovascular finding were not taken into consideration, as it was fairly frequent and was found in a number of unrelated conditions.

We have to depend for the autopsies upon the authorisation by the Coroner in those cases when the patient had died within twenty-four hours of admission into the hospital, or upon the permission of the relatives in those instances when the patient had remained longer in the hospital before death. Often we have difficulty in obtaining the permission of the relatives, more particularly when the case happens to be a Mahomedan, Christian or a Parsi. The same difficulty is experienced with regard to the female bodies. In view of these difficulties, attempts are made by the house staff to get the permission, more often in those cases that are interesting or when the clinical diagnosis is doubtful.

In this series of 4,335 autopsies, major cardiovascular lesions were encountered in 568 or 13·1% of the cases. The clinical records of these cases were also studied and relevant data were taken into consideration.

Of these 568 cases mentioned above 244 (42·95%) had died within the first twentyfour hours of admission into the hospital. Perhaps this high percentage may be accounted for by the difficulty already referred to, in securing autopsies on cases that had remained longer in the hospital. The number of females in this group of cases was 70 (12·3%). This may not represent the true incidence of heart diseases in females, for the reasons mentioned above.

The age distribution of the cases is shown in Table I. In 30 cases the age could not be ascertained from the available records. The number of autopsies done on children in the department is not considerable and this may account for the small number of cases in the first decade. The majority of cases fall between the ages of twenty and fifty years.

TABLE I—AGE DISTRIBUTION OF THE CASES IN THIS SERIES

Age	*No. of cases*
0— 9 years	6
10—19 „	27
20—29 „	91
30—39 „	149
40—49 „	120
50—59 „	97
60 years and over	48
Age not ascertained	30
Total	568

In 279 or 49·1% of the cases, the cardiovascular lesion was only an incidental finding in the course of another disease. In the remaining 289 cases the cardiovascular lesion was responsible for the death of the patient.

The incidence of the various aetiological types is set forth below (Table II).

TABLE II—INCIDENCE OF THE VARIOUS AETIOLOGICAL TYPES *

Aetiology	*No. of cases*	*p.c. of the total*
Congenital lesions	21	3·5
Infective conditions	65	11·1
Rheumatic carditis and valvular lesions	35	6·1
Acute bacterial endocarditis	32	5·6
Subacute bacterial endocarditis	10	1·7
Thrombotic endocarditis	13	2·2
Cardio-aortic syphilis	170	29·3
Hypertension	69	11·9
Athero-sclerosis	56	9·8
Pulmonary heart disease "Cor pulmonale"	45	8·1
Miscellaneous conditions	62	10·7
Total	578	

* No. of times each lesion was encountered.

In a few of the cases lesions due to more than one aetiological factor were encountered and these instances have been included once each under each of the aetiological heads. I could not find in the available literature comparable post-mortem statistics from other places in this country. In Table III is shown our data compared with some recent American data from a paper by Perry and Langsam[8]

TABLE III—COMPARATIVE DATA ABOUT INCIDENCE OF AETIOLOGICAL TYPES GIVEN IN PERCENTAGES

Aetiological Types.	*Present Series*	*Perry & Langsam.* population White	population Negro
Congenital	3·5	1·5	0·9
Inflammatory heart disease†	18·42	19·1	12·7
Pulmonary heart	8·1	1·5	1·6
Arteriosclerotic disease	9·8	32·4	7·1
Hypertensive vascular disease	11·9	33·8	59·1
Syphilitic vascular disease	29·3	4·4	14·2
Rheumatic heart disease	6·2	7·4	3·8

† The acute and subacute bacterial endocarditis has also been included under this head here.

Perry and Langsam include acute and subacute bacterial endocarditis also under inflammatory conditions. Comparing the figures one finds that cardiovascular syphilis forms the single largest group in the present series, whereas hypertensive heart disease forms the single largest group both in White and Negro population there. The incidence of rheumatic heart disease seems to be not very low in our series and is almost equal to the incidence in the White population and is considerably higher than that in the Negro population.

In the subsequent paragraphs I shall discuss in greater detail each of the aetiological groups.

CONGENITAL LESIONS

There were 21 cases of congenital lesions in the present series. Two cases, one of patent ductus arteriosus, and another of patent foramen ovale in infants of one, and one and one half months respectively have not been included. The following table (Table IV) shows the frequency of the different types of lesions encountered.

TABLE IV—CONGENITAL LESIONS

Lesion	*No. of cases*
Patent foramen ovale	17
Bicuspid aortic valve	1
Pulmonary stenosis	2
Undetermined	1
Total	21

The lesion was not directly responsible for the death in any of the cases listed above. In one case, patent foramen ovale was associated with acute bacterial endocarditis of the mitral and aortic valves of undetermined aetiology and in another the condition was associated with hypertensive heart disease.

INFECTIVE CONDITIONS

In this group have been included all the cases of pericarditis, pyogenic as well as tubercular, and myocarditis. The following table (Table V) gives a summary of the data.

TABLE V

Condition	*No. of cases*
Purulent pericarditis following pneumonia	23
Purulent pericarditis associated with other causes	16
Tubercular pericarditis	23
Abscess of the heart wall (embolic)	1
Postdiphtheritic myocarditis	1
Infective myocarditis (undetermined origin.)	1
Total	65

Postpneumonic pericarditis accounts for a little more than one third of the cases and so does tubercular pericarditis. In all these cases except in two of tubercular pericarditis the condition had been unrecognised during life, as it had been masked by the antecedent condition and was detected only on postmortem

examination. In the case of embolic abscess in the heart wall the condition had been masked during life by the causative condition, namely pyæmia.

Rheumatic Cardiac Lesions

In the present series we had 35 cases of chronic rheumatic heart disease. The data are summarised in Table VI.

Table VI—Rheumatic Group

Condition	*No. of cases*
Mitral endocarditis	1
Healed endocarditis of mitral valve without valvular dysfunction.	3
Mitral stenosis alone	18
Mitral stenosis with ulcerative endocarditis ..	3
Mitral stenosis with regurgitation	2
Mitral stenosis with aortic valvular lesions ..	3
Mitral stenosis with aortic syphilis	2
Mitral stenosis with pulmonary stenosis ..	1
Chronic mediastino-pericarditis	2
Total ..	35

Two cases of Pick's syndrome have also been included here, as there was no evidence of any other pathological condition, in these two cases. 4 out of the 35 cases listed above were under twenty years of age, 14 were under thirty and 17 were over thirty, 28 of these cases were males and 7 were females.

It has been the usual teaching so far that acute rheumatic fever and its cardiac complications are rare if not unknown in this country. But a number of reports based upon clinical and autopsy material have been published since 1930 from various parts of the country adducing evidence to disprove this contention. Stott[9] from Lucknow reported a number of cases of mitral stenosis. He held that unrecognised subacute rheumatic infection in childhood to be the cause of mitral stenosis in these cases. Hodges[2] from Bengal reported cases in children with the more uncommon features of juvenile rheumatism. Mangalik and Stott[5] reported that in a series of 118 post-mortem examinations at Lucknow between 1930 and 1936, they had no case of acute rheumatic fever, but found 6 cases (5·1%) of chronic rheumatic heart disease. Stott[10] in another series of 661 autopsies had 15 cases (2·3%) of chronic rheumatic heart disease. 10 of these cases had died of congestive cardiac failure. In our series 18 out of the 35 had died of congestive cardiac failure.

Kutumbiah[3] from Vizagapatam (South India) reported 50 cases of juvenile rheumatism. Kutumbiah[4] was able to demonstrate Aschoff's nodes in sections from the heart of patients dying of rheumatic valvular disease. In our own material even in those instances where search was made for Aschoff's nodes we have not been able to detect them. But sections had not been taken always with standard technique and moreover sufficient number of sections had not always been examined. Inability to demonstrate Aschoff's nodes cannot, however, be taken as positive proof of the case being not one of rheumatic carditis, as they are not always found in every case. From our data it would appear that cardiac rheumatism does form an important cause of heart disease in this part of the country.

Acute, Sub-acute and Thrombotic Endocarditis

This forms a group of 55 cases. The incidence of various forms are shown in Table VII.

Table VII—Acute, subacute, and thrombotic endocarditis

Condition	*No. of cases*
Acute bacterial endocarditis	32
Subacute bacterial endocarditis	10
Terminal verrucous endocarditis	13
Total ..	55

Seven of the cases of acute bacterial endocarditis were a complication of lobar pneumonia. One case was a complication of bacillus pestis septicæmia. In another case the mural endocardium over the site of a cardiac infarct was the seat of involvement, but the causative organism has not been mentioned.

In the 10 cases of subacute bacterial endocarditis, the diagnosis was arrived at from the general clinical and post-mortem picture rather than from the bacteriological findings. Antemortem blood culture was done only in two cases and in those it was found sterile. Post-mortem blood cultures had not been done in these cases.

The remaining 13 cases have been put under terminal verrucous endocarditis. In these cases embolic phenomena were absent and the valve cusps involved were the seat of fairly hard and firm vegetations. The largest of these vegetations was about the size of a cherry and the smallest was about the size of a pin-head. In three of these cases there was associated aortic syphilis. In the others it was found in association with such unrelated conditions as cirrhosis of liver, amœbic colitis with liver abscess and miliary tuberculosis. Unfortunately, sections of these vegetations had not been taken for study.

The frequency with which the different valves were involved is given in Table VIII.

TABLE VIII—ACUTE, SUBACUTE AND THROMBOTIC ENDOCARDITIS

Valve	*No. of cases*
Mitral valve alone	25
Aortic valve alone	14
Tricuspid valve alone	1
Pulmonary valve alone	2
Mural endocardium	2
Aortic and mitral valve	9
Aortic and tricuspid valve	2
Total	55

It was found that the aortic valve had been involved in a considerable number of the cases.

CARDIOVASCULAR SYPHILIS

Excepting one case of syphilitic endarteritis of the pulmonary vessels (Ayerza's disease) and six of aneurysms of the peripheral arterial trunks, this group consists of cases of cardio-aortic syphilis. As I have pointed out above, cardiovascular syphilis forms the single largest group in the present series. The hospital caters mainly to the working and the lower middle classes, and autopsy is not done in every fatal case. On these two points it may be argued that the figures given here do not represent the correct incidence of cardiovascular syphilis, in the community. The problem cannot be settled until accurate figures are available from the clinical services of this and the other hospitals and private clinics in this city. Despite this fact I think the figures given here do give us a fairly accurate estimate of the incidence of cardio-aortic syphilis, for the following reasons. The venereal department of the hospital does claim the largest outpatient attendance, and besides in the clinical services one does see a good number of cases of syphilitic heart disease though exact figures are not at present available. The general proportion of cardiovascular syphilis among all autopsies in this series is no higher than that from cities of other countries (*vide* Table IX).

TABLE IX—INCIDENCE OF CARDIOVASCULAR SYPHILIS (MODIFIED FROM CAREY COOMBS[1])

Authority & Place	*Date*	*No. of autopsies*	*Cardiovas. Syphilis. No. of cases*	*Cardiovas. Syphilis. Percentage.*
Symmers: New York	1916	4,880	175	3·5
Reid: Boston	1920	1,678	54	3·2
Cowan & Foulds: Glasgow	1929	1,000	60	6·0
Taylor: Bristol	1919-29	1,750	45	2·5
Culliman: London	1920-26	1,000	33	3·3
Clauson & Bell: Minneapolis	1910-26	4,577*	126	2·6
Present series: Bombay	1926-39	4,335	170	3·9

* Patients over 20 years.

The frequency of the various lesions encountered is given in Table X.

TABLE X—SYPHILITIC LESIONS

Myocarditis	1
Simple uncomplicated aortitis	55
Aortitis with myocarditis	8
Aortitis with valvular dysfunction	30
Aortitis with valvular dysfunction with myocarditis	3
Aortitis with valvular dysfunction with acute myocardial infarction	1
Aortitis with acute myocardial infarction	8
Gummatous endocarditis of the mitral valve	1
Aneurysm of the heart wall	4
Pulmonary endarteritis	1
Aneurysm of aorta and arterial trunks	58
Total	170

In 32·5% of all cases of (55 in 170) cardiovascular syphilis, there was only minimal lesions, consisting on naked eye examination of longitudinal scarring of the aorta and typical syphilitic meso-aortitis, on microscopical examination. A large number of these cases had died as a result of unrelated conditions. There was one case in which there was gummatous endocarditis of the mitral valve cusps and it is mentioned here, as syphilitic involvement of mitral valve is considered to be rare. The aortic cusps in this case were normal.

Of the 58 aneurysms encountered, six were of the peripheral arterial trunk as mentioned above. One was of the coronary artery and had been reported earlier by Manohar[6]. Two were of the internal carotid artery and one each of right common carotid, left common carotid and left subclavian artery. Three of these five, were associated with aortic syphilis and in two the condition of the aorta was doubtful.

Osler[7] mentions that Leonard Rogers found 30 aneurysms in a series of 5,900 autopsies at the Calcutta Medical College, giving an incidence of 0·05%. The situations of the aneurysms are not mentioned and if we take all the 58 in our series the incidence works out at 1·3%. The frequency with which the different parts of the aorta were found involved in our series is shown in Table XI.

TABLE XI—ANEURYSMS OF THE AORTA

Ascending thoracic aorta	12
Arch of thoracic aorta	11
Descending thoracic aorta	3
Arch, ascending and descending aorta	10
Multiple aneurysms of the thoracic aorta	2
Thoracic aorta (situation not mentioned)	3
Junction of thoracic and abdominal aorta	2
Abdominal aorta	7
Thoracic and abdominal multiple	2
Total	52

Of the seven purely abdominal aneurysm included above, the aneurysmal sac, in one case showed definite syphilitic changes. In the other cases sections from the wall of the aneurysm were not available for study.

Death in 13 of the 58 cases was due to rupture of the wall of the aneurysm. The thoracic aorta was the site of the rupture in eight cases and the abdominal aorta in five cases. The organs in which rupture took place were one each in the pericardial sac, the stomach, the peritoneal cavity and the trachea. In three cases it has been in the retroperitoneal tissues, the left pleura and the œsophagus.

Hypertensive Heart Disease and Athero-Sclerosis

As was mentioned above, cases of atheroma of the aorta, (made out from the naked eye appearance of the aorta in the autopsy room or from a study of sections) without any other cardiovascular lesion have been omitted from this series.

These two groups have been taken together for consideration because cases of hypertension, idiopathic or nephritic in causation also showed atheroma of the aorta, coronary vessels and cardiac valves, either singly or in combination.

There were 69 cases of hypertension and 56 of atherosclerosis. In 45 cases of the former and 35 of the latter the blood pressure had not been mentioned in the clinical records. These cases have been included here because there was definite hypertrophy and dilatation of the heart as was evidenced by considerable enlargement in the size and increase in weight of the heart, above the normal. Besides, there was no other cardiovascular lesion in these cases which could account for the hypertrophy and dilatation of the heart.

The blood pressure recorded clinically was normal in 3 cases in this series under the heading hypertension included for the above mentioned reasons and much above the normal in 4 cases of atherosclerosis. Blood pressure readings have been recorded in each case on a single occasion after admission and some of them have been on patients in a semicomatose condition with renal failure or cerebral hæmorrhage. Whether the readings given represent the true blood pressure of the individual is open to question.

Of the 69 cases of hypertension, 26 were of essential hypertension. The kidneys in 7 of these cases showed changes of arteriosclerotic nephritis. There were 43 cases of nephritic hypertension. The kidneys of these cases showed changes of subacute or chronic nephritis, excepting in one case of bilateral polycystic kidneys and another of bilateral hydronephrosis. Two cases showed adenoma of the adrenal cortical tissue but no endocrine symptoms had been mentioned in the clinical records. In one, blood pressure was 240/110 mms. and in another it was not recorded. In both of these cases the kidneys were normal and, therefore, these two cases have been included under essential hypertension.

The kidneys in 3 cases of atherosclerosis showed a picture of arteriosclerotic nephritis.

The following table (Table XII) shows some of the common findings in the cases of atherosclerosis mentioned above.

Table XII—Atherosclerosis of aorta, coronary vessels and heart valves

Condition	*No. of cases*
Atheroma of aorta and valves without valvular dysfunction	12
Atheroma of aorta and valves producing valvular dysfunction	8
Atheroma of aorta with diffuse dilatation of aorta ..	4
Atheroma of aorta and coronary vessels	28
Aneurysm of heart wall due to coronary sclerosis ..	3
Aneurysm of cœliac artery with atheroma of aorta ..	1
Total ..	56

The cause of death in these cases is shown in Table XIII.

Table XIII

Mode of Death	*Essential Hypertension. No. of cases.*	*Nephritic Hypertension. No. of cases.*	*Atherosclerosis. No. of cases.*
Congestive heart failure ..	3	12	21
Acute myocardial infarction and coronary insufficiency ..	1	0	14
Cerebral hæmorrhage	9	4	3
Uræmia ..	0	12	2
Lobar & Broncho-Pneumonia ..	1	5	3
Peritonitis ..	2	2	2
Traumatic ..	2	2	2
Others ..	6	1	8
Cause not clear from records ..	2	5	1
Total ..	26	43	56

Pulmonary Heart Disease

There were 45 caes of pulmonary heart disease. In all these cases there was definite hypertrophy and dilatation of the heart (as evidenced by increase in weight and enlargement in size of the heart) more particularly of the chambers of the right side. In three

cases there was also syphilitic aortitis. In two cases the condition was associated with chronic nephritis and in another with adherent pericarditis.

One case with considerable enlargement of the heart with extensive emphysema of the lungs was associated by syphilitic aortitis and hypertension.

All the cases, except two of bronchiectasis and two of old healed tuberculosis showed evidence of chronic bronchitis and emphysema of the lungs.

The cause of death in all of these cases was congestive cardiac failure.

Miscellaneous Conditions

This group forms a heterogenous collection of 62 cases. The findings are summarised in the following table.

Table XIV—Miscellaneous Conditions.

Condition	*No. of cases.*
Beriberi heart	3
Malignant deposits in the heart muscle due to melanocarcinoma	3
Malignant infiltration of pericardium	2
Anæmia with congestive heart failure	4
Insufficient data	50
Total ..	62

In fifty of the cases included under miscellaneous conditions though there was evidence of cardiovascular diseases, the clinical as well as the postmortem data were insufficient to assign them to one or the other ætiological group.

Summary and Conclusions

1. The autopsy protocols of 4335 consecutive autopsies done during the period 1926-39, were studied with a view to get data regarding the incidence of the ætiological types of cardiovascular disease.

2. There were 568 (13·1%) cases of cardiovascular disease. 87·7% of these were males.

3. The majority of these cases fell within the age period between 30 and 50 years. This fact may be accounted for by the small number of autopsies on children, and the high incidence of cardiovascular syphilis, in this series.

4. Inflammatory conditions were encountered in 107 (18·4%) cases. The pericardium has been the seat of involvement in 62 (10·1%) cases. Of these 23 were of tubercular pericarditis thus accounting for one third of all cases of pericarditis. The condition has been part of a picture of generalised tuberculosis.

5. Rheumatic heart disease was encountered in 35 (6·2%) cases. The data from available literature as well from our own material supports the contention that rheumatic heart disease is not very uncommon here. Acute, subacute and thrombotic endocarditis account for 55 (9·5%) cases.

6. Cardiovascular syphilis was found in 170 (29·3%) or nearly in one third of the cases. The incidence is no higher than in cities of other countries. Nearly one third of the cases of syphilitic heart disease were of aortitis uncomplicated by valvular dysfunction or aneurysm. Aneurysms accounted for nearly another one-third (58) of the cases of cardiovascular syphilis.

7. There were 126 (21·7%) cases of hypertension and atherosclerosis. Of these 26 were of essential hypertension and 43 were of nephropathic hypertension. Cerebral hæmorrhage was very frequent as the terminating event in the cases of essential hypertension, uræmia in nephropathic hypertension and acute myocardial infarction in cases of atherosclerosis.

8. Pulmonary heart disease was encountered in 45 (8·1%) cases. Seven of these were associated with conditions belonging to other ætiological groups. Death in all these cases was due to congestive cardiac failure.

9. 50 cases could not be assigned to one or the other ætiological group, though they had shown evidence of cardiovascular disease because the available data were insufficient.

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References

1. Coombs (1930)—Leemelian Lectures 1930. *Lancet*, 2: 277, 281, 333.
2. Hodges (1932)—*Indian M. Gaz.*, 67:241.
3. Kutumbiah (1935)—*Indian J. Pediat.*, 2:215.
4. *Idem* (1940)—*Indian J. M. Research*, 22:631.
5. Mangalik and Stott (1938)—*Indian M. Gaz.*, 73:203.
6. Manohar (1938)—*Arch. Path.*, 26:1131.
7. Osler (1909)—Schostein Lectures *Brit. M. J.*, 2:1509.
8. Perry and Langsam (1939)—*Arch. Int. Med.*, 64:971.
9. Stott (1930)—*Indian M. Gaz.*, 65:65.
10. *Idem* (1938)—*Ibid.*, 73:271.

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A SYMPOSIUM ON HIGH BLOOD PRESSURE

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CAPT. P. GANGULI'S ADDRESS

I. HIGH BLOOD PRESSURE

After pulse and respiration, blood pressure gives reasonably exact information regarding life expectancy, from the life history of blood pressure and from mortality data. Hay states that the usual termination of high blood pressure is cardiac defeat. Robert states "the higher the blood pressure, the more we find diseased heart." Apoplexy is the next important complication terminating life. Renal failure is also responsible for death in many cases.

The incidence of high blood pressure is rising in all civilised countries, owing to the increase of the stress and strain of modern life. According to Robinson and Brucer[1] at least 1/5th of population of the United States of America is hypertensive. Death rate is equally high. Fahr[2] states that deaths due to high pressure among persons of 50 years or more was 23 per cent of all deaths during the year 1924. This equals the grand total of all deaths caused by cancer, tuberculosis and all forms of respiratory diseases. According to Statistical Year Book for the German Empire 1927, the deaths caused by cerebral apoplexy alone, which is only one sequel of hypertonia, constituted 5·5 per cent of the total number of deaths. Heart disease accounts for more deaths in the United States than any other two causes and Cabot in his book "Heart and Disease" says that hypertension is more common than all other forms of heart disease put together. Lately Perry and Langsam[3] found in a series of 2066 consecutive autopsies that hypertensive cardiovascular disease was the ætiologic factor in more than half the cases—*viz.*, 55·8 per cent.

II. NORMAL BLOOD PRESSURE—AGE FACTOR

According to Halls Dally[4], blood pressure increases with age. Such great authority as Volhard stated in 1931 that under normal conditions blood pressure increases with advancing years. Dally lays down the following formula for the normal blood pressure.

For ages from 20 to 60, normal systolic blood pressure is 120 plus 1/5th the age so that at the age of 60, the normal systolic pressure is 132 mm. Hg. Then add 1 mm. for each year upto 80 years of age. The normal diastolic pressure is 80 mm. Hg. for 20 years of age. For each five years add 1 mm. Hg. upto 60 years, so that at the age of 60, the normal diastolic pressure is 92 mm. Hg. After 60 years, add 2 mm. Hg. for every 5 years.

In the definition of high blood pressure, he writes that systolic pressure persistently higher than 160 mm. Hg. and diastolic pressure persistently higher than 100 mm. Hg. are to be regarded as high blood pressure. Fritz Lange[5] says that "the maximal blood pressure marking the boundary line of hypertonia is generally placed at 160 mm. Hg." (Romberg 1925). The lower limit of the diastolic pressure is placed at about 100 mm. Hg. The same author says that those statisticians who regard pressure above 170 mm. Hg. as pathologic, aim at low normal values, whereas others are of opinion that figures as high as 210 mm. Hg. are still within physiologic range of variation (Saller[6]).

Many authorities, however, advocate lower values and do not admit that age has anything to do with rise of blood pressure. For instance, Stieglitz[7] considers 140 mm. Hg. and 90 mm. Hg. as the maximal systolic and diastolic pressures, irrespective of age. He says that a systolic of 150 mm. Hg. or more and a diastolic of 95 mm. Hg. or more constitute hypertension. Robinson and Brucer are of opinion that normal systolic blood pressure is from 90 to 120 mm. Hg. and normal diastolic pressure is from 60 to 80 mm. Hg. He adds that a normal person attains mature blood pressure at about adolescence and keeps that range throughout life. Normal blood pressure does not rise with age. If it shows rise, then the person is either a prehypertensive or actual hypertensive subject. Daily and yearly variation of 5 to 10 mm. only can be allowed. In conclusion, Robinson and Brucer say that a blood pressure history of over 120 systolic and 80 diastolic over a 10 years' span in a man or woman is pathologic and is an almost infallible sign of incipient hypertension.

III. STANDARDISATION OF METHODS OF MEASURING ARTERIAL BLOOD PRESSURE

In October 1940, Treloar[8] admitted that the conclusions of Robinson and Brucer about the normal blood pressure and its invariability in spite of the

advance in age may be reasonably sound, although he objected to their methods of presenting statistical manipulation as proof of validity of their conclusions. It is well recognised in biostatics that any attempt to fix limits of normal variation in a physiological function is extremely hazardous. Again, it has been freequently found that the record of the heights of pressure of the same person varies with different technicians and with different instruments and with sizes of armlet or cuffs. The method of taking blood pressure varied a great deal in different countries. In 1938 the American Heart Association appointed a Committee which in collaboration with Cardiac Society of Great Britain issued a joint report in 1939. There were only three points in which agreement was not reached. They were—

(1) The Americans prefer sitting posture during taking blood pressure. The British prefer sitting or lying down, so long as the patient is comfortable.

(2) British prefer mercurial manometer. The Americans make no distinction between mercurial and aneroid instruments, provided they are in good condition.

(3) Both Committees regard diastolic pressure in which sudden muffling of Korotkoff's sounds occur. The Americans recommend that the disappearance of sound should also be recorded like 80-70, 40-0, 80-80 and so on.

Points of Agreement:—Cuff should be at least 12 c.m. wide applied one inch above the bend of the elbow.

Some Important Points—(1) Pressure of both arms should be taken. (2) In all cases of high blood pressure, pressure of the leg should be taken to exclude coarctation of aorta. (3) Blood pressure should be taken in the morning, 10 to 12 hours after the last night meal, after resting in a warm room for half hour.

Graphic Methods:—Pachon and Fabre[9] have introduced graphic methods, in which the personal factor of error is totally abolished. They are tensiograph and oscillograph in which the blood pressure is recorded by graphs. Both these instruments should be calibrated by comparing with mercurial manometer. A few graphs of interesting cases are reproduced later.

IV. Potential Hypertensives

It has become possible to detect potential hypertensives by several tests, the main principle of which depends on vascular function of each individual. (*a*) Hine's[10] readings of 1185 persons 10 to 20 years after initial reading showed that those with elevation of blood pressure on the original readings subsequently developed hypertension. He also found that instability of blood pressure is also a precursor of hypertension. (*b*) Cold Pressor test of Hines and Brown[11]:—The subject sits in a warm room for 20 to 60 minutes. The cuff of the sphygmomanometer is applied in one arm and the other hand is immersed in icecold water (4°C) upto just above the wrist. With the hand still in water, readings are taken at the end of 30 and 60 seconds. After 60 seconds, the hand is removed. Readings are taken every two minutes until pressure returns to normal. If the pressure returns to normal within 2 minutes, the man is normal, but if it is delayed, he is hypertensive. Also a rise of 20 systolic and 15 diastolic or more pressure indicates hyperactivity. A maximal rise above 140 systolic and 90 disastolic pressure is also a definite evidence of hyper-reactivity, which indicates potential hypertonic.

(*c*) Allam and Smirk[12] employed cold pressure test with immersion of 2/3rd of forearm and arrest of circulation. The blood pressure rises. In essential hypertension, there was larger rise of pressure than in normal persons, but in hypertension of chronic nephritis, the rise in both systolic and diastolic pressures was less than in even normal subjects.

(*d*) The same workers found that if ischæmic arm is exercised there is no change or slight slowing of pulse rate in normal persons. The rate goes up in hypertension.

(*e*) Lange's[5] afterflow test in nail capillaries: If the flow of blood in arteries is stopped by inflating the cuff of a sphygmomanometer, considerable time elapses before the flow of blood comes to a standstill in nail capillaries. This time is constant in one and the same individual. In normal persons the afterflow becomes shorter under the influence of heat and longer under influence of cold. In hypertonic patients, the afterflow is longer under the influence of heat and shorter under cold. In arteriosclerotics, the effects of heat and cold are the same.

All these blood pressure rising tests are indicators of hyper-reactive states of vasomotor mechanism rather than an essential hypertension, but they may be regarded as precursor of actual change which results in high blood pressure.

V. Classification of High Blood Pressure

The main classification is renal and non-renal hypertension. Blood pressure is raised in various

primary renal diseases of which the most important are acute and chronic glomerular nephritis, pyelonephritis and polycystic renal diseases. Among nephrosis, severe amyloidosis of kidney is associated with high blood pressure. All varieties of urinary obstruction causes rise in blood pressure. Any condition which diminishes blood supply to the kidneys, such as arteriosclerosis and periarterial nodosa of renal vessels may cause rise of blood pressure. Goldblatt's classic clamping experiments of renal arteries[13] of dogs opened up a possible explanation that an ischæmic condition of the kidneys due to obstruction or reduction of flow of blood to the kidneys is the cause of rise of blood pressure and it has been thought that essential hypertension is of renal origin due to the same cause. Goldblatt's experiments have been corroborated by many workers in different ways and manners. For example, Drury[14] adopted a new method of putting a ligature round the left renal artery in a baby rabbit. As it grew, the left kidney dwarfed, and an increase in arterial tension resulted in spite of hypertrophy of the right kidney. This high tension was lowered where the left ischæmic kidney was removed. Ryland[15] produced hypertension in rats by partial occlusion of aorta which disappears after nephrectomy. High blood pressure in coarctation of aorta is attributed to reduction of blood flow into the kidneys for if coarctation occurs below the origin of the renal vessels, no rise of blood pressure occurs. Again, hypertension in eclampsia has been attributed to ischaemia of the kidneys but there are other explanations. By constriction of renal artery Wilson and Pickering[16] produced actual arterial lesions in rabbits identical with those of malignant hypertension. At the same time Goldbatt[17] produced experimental malignant hypertension.

It has been assumed that a hypothetical pressor substance is liberated by ischaemic kidneys, which raises the blood pressure by producing general vasoconstriction. This is known as Edris Jone's pressor principle. But this pressor substance could not be isolated from blood, although it has been detected in small quantities in urine. Transfusion of blood from hypertonic to anæmic subjects did not produce increase in tension in the receiver. Even cross transfusion experiments of Friedman and others[18] on patients with malignant hypertension and normal persons failed to show any evidence of significant change in blood pressure. It has been suggested that the pressor substances are absorbed by cellular contents of the blood or fixed by endothelial cells of the arteries.

Recently Hessel[19] demonstrated a pressor substance in blood, obtained from the renal vein of a dog which was clamped. If this experimental finding is accepted then essential hypertension can be classed under renal hypertension. Again, extract of ischæmic kidneys produces rise of pressure but that of normal one does not raise it as found by Prinzmetal[20] and others.

It has however been found by Prinzmetal and others that when acetylbeta-methyl-choline was injected subcutaneously into hypertensive subjects, there was a significant fall of blood pressure. But when this experiment was repeated in dogs, no such fall was seen. Engle and Binger[21] consider that the usual type of essential hypertension in man is of a different type from that of renal ischæmia and they suggested that deficient acetylcholine vasodilator mechanism causes essential hypertension.

This finding brings us to consideration of the non-renal causes of hypertension. Scott [22] puts two main nonrenal causes for producing hypertension (1) Endocrine disturbances and (2) Vaso-motor disorders. Under endocrine disturbances he puts (*a*) Basophilic adenoma of the pituitary (*b*) adrenal tumour (*c*) hyperthyroidism (*d*) menopause and (*e*) obesity. Under vasomotor disorders he puts (*a*) increased intracranial pressures (*b*) psychic disturbances (*c*) circulatory failure and (*d*) complete heart block. Schroder and Steel[24] consider both renal and non-renal types of hypertension as secondary hypertensions, which are present in small proportion of cases in which diastolic pressure is elevated. When these are relieved, hypertension often disappears in these secondary ones. He puts these secondary hypertension under five systems :— (1) Genitourinary (2) nervous (3) endocrine and (4) vascular systems and (5) unclassified. The genitourinary diseases have already been mentioned. Under nervous, he puts (*a*) tumours of brain and other lesions of brain giving rise to increased intracranial pressure (*b*) Diseases of brain stem (bulbar polyomyelitis etc.) Under endocrine system, he mentions, besides pituitary and adrenalin tumours, tumours of the ovary and ovarian hypofunction. Under vascular system, he puts (1) obstructive arteriosclerosis of renal artery (2) Periarteriolonodosa of renal artery (3) Coarctation of aorta and (4) lead poisoning.

When none of these diseases is present, an independent disease called essential hypertension is regarded as responsible for the pathological processes observed.

VI. Essential Hypertension

Essential hypertension is present in 85 per cent of all cases in which blood pressure is raised. In most instances the so-called essential hypertension is of renal origin, but this has been questioned by others. Fishberg [23] states that essential hypertension is merely a collective concept—for a number of conditions having in common positive characteristic of arterial hypertension and the negative one of the absence of primary renal disease. Essential hypertension is now-a-days regarded as a syndrome for which any number of different diseases, besides renal ischæmia can be responsible. For instance, Schroder and Steel[24] describe four syndromes of hypertension secondary to renal, nervous, vascular and endocrine disorders. They state that the disturbances associated with essential hypertension can for the most part, be divided into the same four groups. There still remain a few cases, in which both benign and malignant syndromes fall into an unclassified group. They divide essential hypertension into (1) renal hypertension without failure that is to say, in which renal function is intact, (2) nervous hypertension, in which hypertensive diencephalic syndrome preponderates, (3) endocrine hypertension, in which is grouped (*a*) non-goiterous thyrotoxic hypertension and (*b*) menopausal hypertension and such others and (4) arteriosclerotic hypertension, in which it is supposed that renal arteries are involved. He says that essential hypertension is not a primary disease as is ordinarily believed. The fact that this diagnosis is made by exclusion should indicate multiple causes. Boyd[25] says that cerebral lesions may cause hypertension by damaging vasodilator centre in medulla. The difference found among groups of cases is evidence that many causes are possible. In too many instances, the associated disturbances may be significant. The unity of essential hypertension as advocated by Boyd and others is, therefore seriously called into question. Regarding etiology of essential hypertension, East and Baine[26] said that its causation is still obscure. A number of possible reasons for it may be discussed, but none of them are constant in all cases. It is possible that more than one cause may operate. In the light of Schreder and Steel's cases the need of a new classification is obvious. The new classification of essential hypertension as advocated by Schreder and Steele has already been of advantage in facilitating diagnosis with reference to different syndrome under each head, in making prognosis more accurate and in setting the indications for therapy on more reasonable basis.

Essential hypertension is varied in character. The patient may live a long and useful life without any trouble and a pressure above 200 mm. Hg. is compatible with vigorous existence, but sooner or later the patient is likely to die of cerebral hæmorrhage or cardiac failure. In time, signs of renal insufficiency may appear, particularly loss in concentrating power of the kidney. In a small group of cases, these signs develop early and the chief danger is death from renal insufficiency. This group is known as *malignant* hypertension in distinction to the usual *benign* hypertension in which there is little or no evidence of renal failure. The corresponding renal lesions are known as benign or malignant nephrosclerosis. There are various causes of malignant type of essential hypertension of which the renal one is supposed to be most frequent. Non-goiterous toxic hypertonia is essentially malignant.

VII. Genesis of High Blood Pressure

I have already mentioned about E. Idris Jones' pressor principles derived from ischæmic kidneys causing general vasoconstriction and hyperpiesia. The other factors, *viz.*, increased cardiac output and increased viscosity of the blood have been excluded by direct measurement. In normal persons the tonus of muscular layers of the vessels is maintained by the action of vasomotor nerves, and a fall in blood pressure results after destruction of spinal cord and vasodilatation occurs after sympathectomy. But complete vasodilatation does not follow denervation of vessels, hence it is agreed that there is also a chemical factor in maintaining the vascular tone. It has been argued that hypertension may be caused by excessive vasomotor nervous tone which is the result of overactivity of vasomotor centre in the medulla. This is attributed to diminished blood supply to medulla, but this hypothesis had to be given up as in ischæmia of the medulla, respiration ought to have been increased in frequency and depth which is not seen in any case of high blood pressure. Again the supporters of vasomotor genesis pointed out that the hypertensives react abnormally to cold pressor test, so that instead of a moderate rise of blood pressure in normal beings, an unusual rise of blood pressure in hypertonics raises the question of the enhanced irritability of vasomotor centre, but according to Pickering[27] the same phenomenon may be due to increased irritability of the peripheral vessels. Pickering and Kissin[28]

found negative findings of "cold pressure test" in a few cases of essential hypertension. Pickering considers that only chemical factor is responsible for general vasoconstriction and rise of blood pressure.

Katz and Leiter[29] agreed with most of the writers in the relationship of increased peripheral resistance to increased arterial pressure but they believed that the nervous control of the peripheral vessel is more important than the physical and chemical influences, which control the peripheral arterioles. The controlling influence of blood pressure is normally exerted by the proprioceptive mechanism which is also known as moderator mechanism. The peripheral resistance, they say, is mediated locally in the arterioles by way of axon reflex or through the peripheral autonomic ganglia, the dorsal root ganglia or the sympathetic and parasympathetic systems in general. Correlating such effective pathways are the vasomotor nerve system of the spinal cord, medulla and higher centre of the brain. Both humeral and nervous influences may effect initiation of response in these centres. Impulses arrive along the afferent nerves, from the special sense organs, viscera and cardiovascular structures themselves. They may be mediated by any part of the cardiovascular system because, according to Katz and Leiter, the vascular end organs are not limited to carotid arteries and the aorta only, but are found rather widely distributed in the large vessels. The most important end organs are located in the carotid sinus. These and the associated nerve supply constitute the moderator mechanism and play a most important part in normal control of blood pressure. Hypertension they say, may result from one of the following causes. Due to arteriosclerosis or loss of elasticity of the vessel walls, the response of the moderator mechanism may be much reduced with loss of ability to control the effects of ordinary stimulation, or there may be successive stresses on the normal moderator mechanism exceeding the ability of this mechanism to cope with them. It is stated that an inherent weakness of this mechanism is its ineffectiveness in response to a chronic, persistent elevation of pressure. An abrupt transitory stimulus to the moderator is more effective. Katz and Leiter contend that all types of hypertension are results of disproportion between strain on the moderator mechanism and its ability to cope with such stresses. In case of essential hypertension, they are of opinion that the peripheral vascular narrowing is universally distributed and this may be due to the excessive strain on the moderator mechanism from stimulation of the vasomotor apparatus arriving from sensorium and higher centres of brain or by disease or disturbance of blood supply to the centres. Such strains on moderator mechanism may also be effected by humoral factor such as observed in hyperplasia or tumour of the adrenal cortex.

There is, however, no evidence to prove that these substances arising from the hypophysis or from the adrenal cortex or from the kidney which produce sustained elevation of blood pressure are mediated by this mechanism. The operations involving the removal of sympathetic plexus around femoral arteries or the section of the anterior spinal roots from the 6th thoracic to the 2nd lumbar and section of the major and minor splanchnic nerves above and below the diaphragm with removal of adjacent sympathetic ganglia, have in some cases reduced the blood pressure of hypertensive patients and in some cases, the reduction has been permanent. In many cases, however, the fall was only temporary and hence the operation was unproductive of any tangible result. In 1935, Pickering[27] suggested an experiment, by which he conclusively proved that the genesis of hypertension was chemical and not nervous. The experiment depended on the principle that the blood flow is proportional to perfusing pressure divided by the resistance offered by the vessel. If the genesis is nervous, then in hypertensive subjects, blood flow should be increased within the arteries from which vasomotor nervous control is removed, which can be easily done in one of the extremities by raising the body temperature. The actual experiments however proved that the blood flow was not increased in hypertensive subjects, disproving the vasomotor nervous origin of general vasoconstriction responsible for raising the blood pressure. This is one of the strong proofs of the chemical origin of vasoconstriction. This phenomenon was observed in essential hypertension, both benign and malignant, as well as that associated with chronic nephritis. Pickering found the same phenomenon in high blood pressure due to aortic coarctation. There was, however, one exception. In acute nephritis associated with hypertension, there was increased flow of blood through arteries of the hand, from which vasomotor tone had been abolished by raising the body temperature, showing that vasoconstriction in acute nephritis is due to nervous origin.

These observations suggest that although vasomotor nervous system may control local regulation of blood flow by producing vasorelaxation or vasocons-

triction according to the needs of those particular parts of the body, general vasoconstriction, which is responsible for high blood pressure, is chiefly the result of some chemical or homonic pressor substance generated probably in the cortex of the ischæmic kidneys or some other parts of the body.

Jacobson[30] observes that it is among the neuromuscular hypertensives that "essential" hypertension is distributed and he suggests that essential hypertensive patients should be examined systematically for signs of this condition, with the help of electromyogram. In a normal individual, the string remains quiet, but in neuromuscular hypertensives the string vibrates on account of "action potentials", to the extent of 1 mv. or more. In these cases, increase of blood pressure is attributed to generalised increase of skeletal and visceral muscular tension, definitely measurable in action potentials (plus the brain and cord and chemical processes inevitably involved).

VIII. Anatomical Changes in Hypertension with Reference to Symptoms

The conditions which may be responsible for death in hypertension are mainly heart failure, apoplexy and kidney failure. Heart failure occurs in 60 per cent of all cases with hypertension (Bell[31]). As a rule failure develops with a normal rhythm, so that pulse or heart sound does not give any indication of heart failure. Extra systoles of ventricles are common, even though the rate is fast. A few cases develop auricular fibrillation. The left ventricle may fail without any conspicuous morbid changes being found in it at post mortem. In some cases, myocardial fibrosis of fine and patchy distribution of the fibres due to small coronary arteriolar infarcts is found. Sometimes there are gross arteriosclerotic changes of the coronary arteries. In 30 per cent of Levine's[32] series of coronary thrombosis, there was hypertension. The heart failure may occur from exhaustion alone and this is accelerated if coronary vessels suffer from arteriosclerosis. In hypertrophy of the left ventricle, relative coronary insufficiency is the result in spite of normal size of the coronary arterial calibre.

In massive hæmorrhages two factors are associated, *viz.*, arterial degeneration and high blood pressure. The softening of the supporting brain tissue, through narrowing or thrombosis of the blood vessels from any cause may lead to hæmorrhage in high blood pressure.

Hypertensive encephalopathy (Fishberg and Oppenheimer[33]) is the name given to hypertensive cerebral attacks which occur in essential as well as nephritic hypertension. The symptoms are caused by a rapid rise in blood pressure above the previous level. McAlpine[34] distinguishes two forms of attack; the basis of one is cerebral œdema and that of the other is angiospasm. Both cause cerebral anæmia. Ricker[35] found as a result of animal experiments, that when an artery became extremely contracted by spasm or otherwise its capillaries became markedly dilated with stasis of the blood stream followed by diapedesis of red blood cells through the capillary walls. The contraction of the vessel leads to ischæmic necrosis of the wall of the distal segment, so that when spasm passes off and the blood again flows through the vessel, either diapedesis or actual hæmorrhage may occur. What appears to be massive hæmorrhage may consist in reality of a large number of these small hæmorrhages. Westphal and Bär[36] describe the condition as angionecrosis.

In a very large number of cases however, especially in elderly people who have got atheroma of cerebral vessels or syphilitic endarteritis, thrombosis may occur, during lowering of blood pressure either from a natural or artificial cause. Most frequently it is the result of injudicious administration of hypotensive drugs.

It has been seen that persons suffering from hypertonia exhibit a tendency to local spasms as shown by sudden whitening of the fingers and sudden onsets of vascular spasms. Pickering[27] considers that there cannot be local spasms unless the local vessels are diseased. In essential hypertension there is a functional increase in the tonus or excitability and the hypertrophic anatomical changes in the media and hyperplastic sclerosis of the arterioles are secondary. The constant peculiarity of hypertonic blood vessels is that they become unusually responsive to stimulation. It is due to hyperexcitability that hypertonic arteries are subjected by physiological stimuli to greater and to more varied alterations in their width than normal arteries. This spasm is so much increased in malignant type of hypertension that it leads to arteriolonecrosis in the vessels of the kidneys and of the brain including the retinal arteries. A person suffering from benign essential hypertension turns pale or blushes; he complains of blood rushing to his head; he suffers from palpitation and in fact everything indicates the frequency of intensity of alternating vascular dilatation and contraction. This implies greater amount of stress on arterial

walls, which leads to arteriosclerotic and hypertrophic changes in media, differing in different parts of the body. The hypertrophic changes are most marked in skeletal muscles and skin. Blood vessels of arteriosclerosis and hypertension react differently to stimuli. This has been proved by Lange's "after-flow test" already described.

Hypertensive encephalopathy is chiefly due to cerebral angiospasms leading to temporary ischæmia of the brain, which according to severity, lead to various symptoms, ranging from reeling or other forms of transient disturbances of functions to cerebral hæmorrhage. In some fatal cases, nothing abnormal was detected in the brain, who die of hypertensive encephalopathy just as in some fatal cases of angina pectoris, no coronary thrombosis is detected, when post mortem examination is done. Cerebral asthma is supposed to be caused by sudden attacks of vaso-constriction in the respiratory centre. Intermittent claudication, temporary disturbance of vision or amaurosis are all attributable to sudden vasoconstriction. Hypertensive encephalopathy is usually a complication of essential hypertension, hypertension of chronic glomerular nephritis, eclampsia and acute lead poisoning. That which is due to cerebral œdema will produce cerebral anæmia, bilateral amaurosis, coma and convulsions. Intracranial pressure is raised causing severe headache. In the other form, when anæmia is the result of simple vasoconstriction, pial and meningeal vessels are chiefly involved. The result is also convulsion and coma. Although amaurosis, temporary paresis and paralysis are met with in hypertensive encephalopathy, typical persistent hemiplegia is seldom met with, thus distinguishing it from true apoplexy. Besides in apoplexy, convulsions are uncommon in shock stage, reflexes are lost, but in hypertensive encephalopathy, plantar reflex is increased. It can be distinguished from true uræmia in which there is no sudden rise of blood pressure and no true convulsions, although muscular twitchings are frequently seen especially in children suffering from acute glomerulonephritis associated with high blood pressure and also with high cerebrospinal pressure. True uræmia comes on as a complication of chronic glomerulonephritis and malignant type of essential hypertension.

The anatomical changes always occur in the brain and retinal vessels of hypertensives, as the case goes on. Arteriosclerosis commonly goes hand in hand with hypertension, but in many cases hypertension occurs without any retinal arteriosclerosis. Here are included cases of great diversity, such as essential hypertension, acute nephritis, mild toxæmias of pregnancy, increased intracranial pressure, cardiac decompensation with transitory hypertension, hyperthyroidism with high systolic pressure and others. In these cases nothing abnormal is found in retina. In essential hypertension, arteriolosclerosis gradually takes place in cerebral vessels and this is manifested in the retina by greater visibility of arterioles in the macular and peripheral regions of the eye ground. The lumina of the arteries become also gradually constricted with the progress of the disease, the branches arise at acute angles, arteriovenous constriction becomes more marked and fine variations of calibre are almost always seen. With the development of arteriosclerosis, light streaks, hæmorrhages and exudations or white spots appear in advanced cases. Finally, it becomes hard to distinguish it from albuminuric retinitis of glomerular nephritis. In malignant types of hypertension, papillœdema is usually first manifested before neuroretinitis comes on, with soft, cotton-wool like 'white' spots, although 'hard' spots of arteriosclerotic retinitis may also be present. The lumen of the arteries are extremely narrowed. This condition may also be found in hypertensive toxæmia of pregnancy and lead poisoning with hypertension. Robert U Gillan[37], in a review of 83 cases, described gradient decrease in the lumen of the arteries from first to fifth degree. The fifth degree in which thread like appearance of blood stream is visible in one or more arteries indicate proneness to vascular lesion in the brain. He, however, found that none of the six cases in that series which showed retinal hæmorrhages died or were subject of cerebral hæmorrhage up to the time of his report. Opinions vary in this point, as in malignant variety of essential hypertension, hæmorrhages and lesions in the brain are most abundant owing to arteriolonecrosis, but death occurs so quickly from renal failure that small cerebral hæmorrhages are not manifested by apoplexy. A clinicopathological study of the brain of 12 cases of malignant hypertension by Edward F. Rosenberg[38] in which the duration of life after the onset of symptoms varied from four to eighteen months and averaged eight and nineteenths months. In all of them, œdema of the optic discs was present in life. He found that the brain was seriously damaged in all cases. No vital organ save the kidney was more uniformly or more extensively damaged by this disease. His conclusions were: (1) The cerebral arterioles

were profoundly altered. The alteration took the form of an increase in the thickness of the walls with reduction of the calibre of the lumen. (2) The brain was frequently seriously injured by vascular lesions. (3) Patients with malignant hypertension whose clinical course suggested that cerebral injury had occurred can be divided into distinct groups on the basis of the nature of these symptoms, and for each group a characteristic pathologic picture can be inferred with considerable accuracy. (4) Transcient cerebral phenomena of malignant hypertension that have previously been designated by terms such as "cerebral crisis" or "hypertensive encephalopathy" and ascribed to 'vasospasm' may often be associated with widespread destructive lesions.

Among a number of important papers by Wagner and Keith,[39] Williams and Harrison,[40] Keith, Wagner and Barker[41], Wesselow and Thomson,[42] Page,[43] Wilson and Byron,[44] Rosenthal and Lander[45] the monograph of Wagner and Keith is worth special notice. They lay great stress on the retinopathy in classification between benign and malignant hypertension and state that the lesion in the retinal arterioles is essentially anatomic resulting either from previous angiospastic or from long continued increase in arteriolar tonus without active angiospastic episodes. In severe or malignant hypertension, the essential lesion in the arterioles is active spastic constriction superimposed either on increased arteriolar tonus or on anatomic thickening of the walls of the arterioles or on both.

Relation of Symptoms to Lesions—In 1928 Oppenheimer and Fishberg[33], reviewed the subject of acute episodes of cerebral phenomenon that occur in association with various hypertensive states and independent of uræmia. They stated that examination of brain of a patient who died after having had these acute manifestations does not reveal any focal lesions to account for the epileptiform convulsions and other symptoms such as coma, headache, amaurosis, hemiplegia and aphasia, which are so striking during life. Such brains, they stated, show only inconstant œdema or anæmia. They concluded that the syndrome is primarily due to hypertension and that vasospasms were directly responsible.

So far for the benign hypertension. But in malignant hypertensions, it was always possible to demonstrate regions of cerebral destruction when focal cerebral symptoms had been present during life. Davidson and Brill[46] found that when focal cerebral symptoms were present, destructive lesions in the brain are also found.

Rosenberg[38] proposed clinicopathologic classification of these cerebral phenomena of malignant hypertension as follows: Three primary symptoms which occur in such conditions are: (1) symptoms of increased intracranial pressure, (2) symptoms of mutiple miliary cerebral lesions and (3) symptoms of large cerebrovascular accidents. Increased intracranial pressure results from intracranial and extracranial œdema and include severe headaches, nausea, vomiting, mental dullness and drowsiness. In these cases, no destructive lesions of brain tissue can be found. Only arteriolar thickening and narrowing of the lumen of the vessels may be found. When the brain has suffered widespread multiple infarctions or hæmorrhages or both, a wide variety of symptoms result. The region of the brain, which is injured often, is not large enough to cause disturbance in reflexes or in sensations or motor power and the results of neurological examinations are usually negative. Episodes of vertigo and staggering suggest damage to the equilibratory system. When olfactory hallucinations occur, a region of the hippocampus is suggested to be injured. Transient hemiplegias and aphasias suggest lesions in the great projection systems of the cortex. Changes in personality suggest damage to the frontal lobe. The observation of many small cortical lesions in autopsy provided an explanation for the occurrence of convulsions in some of these cases. Rosenberg cites a case of a man aged 50 years with severe hypertension and heart failure. During the last months of his life, he had convulsions, periods of coma and of marked restlessness and personality changes followed by delirium and terminal coma. Autopsy revealed multiple lesions in the brain due to hæmorrhages and infarcts. Large cerebral hæmorrhage may occur after preliminary multiple focal lesions or such multiple destructive lesions may develop fatal œdema of the brain in the form of signs of increased intracranial pressure. Rosenberg cites another case in which no evidence of paralysis or confusions were noticed during life, but severe symptoms of increased intracranial pressure and cerebral damage appeared in the form of violent headache, mental dullness, olfactory, visual and auditory hallucinations and finally confusion, disturbing thoughts, terminal stupor and coma. At autopsy, arteriolar changes were only moderate in the cortex and in the basal ganglia and brain stem. But the brain was unusually heavy owing to œdema and gliosis of the cerebrum. An indeterminate cerebral

substance was entirely destroyed by small macroscopic and countless microscopic infarctions of varying ages.

In the past, many of these symptoms occurring in patients were attributed to spasm of cerebral vessels. These spasms may indeed occur and perhaps are responsible for the lesions. However, such an assumption may lead the clinician to an unjustified sense of security concerning the brain. Gustave Ricker's[35] observations suggest that severe vascular spasms are attended with hæmorrhages and when large number of branches are involved, extensive hæmorrhages can occur.

In 1935, Page[47] described a symptom complex occurring in young women suffering from essential hypertension, which he called the "hypertensive diencephalic syndrome." They consist of (1) curious, irregular, blochy blushing, first of face then spreading to the neck, the trunk and sometimes the abdomen; (2) spontaneous crying or watering of the eyes; (3) excessive perspiration often at the site of the blush; (4) palpitation of the heart and increase in the cardiac rate (5) rise in blood pressure and (6) coldness of the extremities. Attacks are usually brought on by emotional stimuli, but may arise spontaneously.

Clinical Manifestations of High Blood-Pressure in Renal Diseases—In case of chronic glomerulonephritis, the kidneys gradually undergo nephrosclerosis from the stage of nephrotic nephritis and is gradually associated with failure of elimination of nitrogenous metabolic products, polyurea and rise of blood pressure. The end is usually uræmia, unless the patient is previously carried off by some intercurrent disease. Pyelonephritis also produces hypertension in the terminal stages, and that 15 to 20 per cent of all cases of malignant hypertension can be attributed to it. The symptoms are commonly those of uræmia rather than those of severe coronary or cerebral arteriosclerosis. Ashton Graybiel[48] after a study of 100 selected cases found that cerebral encephalopathy, neuroretinitis and high cerebral fluid pressure as well as the syndrome of left ventricular failure were frequently observed. In case of glomerulonephritis, hypertension gradually comes on after the subacute stage passes on to the chronic one, the glomeruli become gradually degenerated cutting off the blood supply to the tubules initiating degenerative changes in them. Later on scarring takes place, contracting the kidneys, which become shrivelled up and the capsules become firmly adherent to the cortex, which is extremely shrunken. Islands of regeneration appear alternating irregularly with patches of sclerosed tissue like those in cirrhosis of the liver. The convulted tubules show the most advanced degree of atrophy, corresponding exactly with that of glomeruli. Some escape atrophy owing to enlargement of Ludwig's vessel and collateral circulation being established from that source. The blood vessels show at first diffuse hyperplastic sclerosis characterized by hyalinsation of the intima and splitting of the internal elastic lamina like that of essential hypertension followed by endarteritis obliterans, which is partly inflammatory in origin. As scarring proceeds the blood pressure rises. This rise of blood pressure is also seen in acute stage, which, as has already been stated is partly nervous in origin. The most important factor for filtration of urine is the pressure of the circulating blood, and in order to overcome the difficulty in the process of filtration of urine a beneficient compensatory reaction takes place in the shape of hypertrophy of the left ventricle and rise of blood pressure. The hyperplastic thickening of the intima which occurs at first, is regarded as secondary to high blood pressure, just as in the kidneys of essential hypertension. The eye ground shows typical albuminuric retinitis characterised by exudates in the shape of "cotton wool" patches and stars in the macula and also superficial 'flameshaped' hæmorrhages. Urea clearance test shows impairment of function of eliminating urea and blood urea rises indicating retention of nonprotein nitrogen and creatinine in the blood due to defect of the glomerular filtration apparatus. When this occurs dyspnœa becomes sometimes a prominent symptom owing to retention of nonvolatile acids by the kidneys. The patient usually dies of uræmia within two years after the onset of high blood pressure.

The kidneys of benign essential hypertension gradually become sclerosed and contracted as a result of vascular degeneration chiefly affecting the small arteries and arterioles in the form of hyperplastic fibrosis of the intima. The same changes occur in the vessels of brain, retina, spleen and pancreas. The effect upon the kidney depends entirely on the extent and intensity of the vascular lesion and on the rapidity with which they develop. In the great majority of cases which are of benign type the kidneys take long time to undergo advanced picture of shrunken arteriosclerotic kidneys with irregular atrophy of the cortex.

The most characteristic finding is a peculiar hyaline thickening of the subendothelial layer of the intima in small arteries, particularly in the vasa afferentia of the glomerules, the effect of which may be completely to

close the lumen, resulting in atrophy of the glomerule and great increase in the interstitial tissue.

In a small proportion of cases (10 to 15 per cent), symptoms of renal insufficiency rapidly develop and the disease persues a malignant course ending usually in uræmia. The renal changes have been described as malignant nephrosclerosis. These cases make rapid downhill progress and there is early impairment of cardiac, renal and retinal function. The blood pressure is much higher than benign. The kidneys are smooth and of normal size for the internal degenerative process, arteriolonecrosis progresses so rapidly and death occurs so swiftly that there is no time for much atrophy to develop. Owing to arteriolonecrosis of the afferent vessels of the kidneys, hæmorrhages occur into glomerular spaces and surrounding tissues, causing epithelial crescents of Volhard to form by exciting proliferation of the capsular endothelium. This also explains subcapsular hæmorrhages giving the kidney "flea-bitten" appearance of embolic glomerulonephritis. The thickening of the media of the vessels within pectoral muscles becomes much more marked in malignant hypertension than anything else, thus distinguishing it from other types of hypertension as worked out by Kernohan, Anderson and Keith.[49] Gustave Ricker's experimental findings which have been already referred to, explain the after-effects of arteriolonecrosis and spasmodic constriction of arterioles not only in the kidneys, but also the lesions in the heart, brain and in the retina as already described. The papilloedema or choked disc of malignant hypertension is the result of cerebral œdema giving rise to increased intracranial tension.

IX. Prognosis

Prof. John A. Ryle[50] prefers the term hyperpiesia to hypertension which has been generally accepted in America. He considers that in each individual case, age, sex, weight, occupation, temperament, mode of life, family history symptoms and physical findings—particularly those relating to the number and quality of the secondary consequences of sustained high pressure and arterial change—must all be taken into account in framing a prognosis. The menopausal and the so-called malignant cases require a separate assessment. But the most noteworthy variations in prognosis are naturally those which accompany the several stages of the disease. Of such stages there may be said to be five.

"In the *first stage* the patient, a man of perhaps 45-50, presents himself without symptoms for a life-assurance examination or a routine overhaul, or for some separate indisposition. He is found to have a blood-pressure, say, of 170/100. Arterial thickening and cardiac hypertrophy cannot be clinically demonstrated and there is no albuminuria. There is hyperpiesia without arteriosclerosis. In this stage we may even conceive that the condition is modifiable. The patient may well have ten, fifteen, or more years without disability."

"In the *second stage* he presents himself for slight symptoms often referable to head or heart and commencing signs of secondary pathology. There may be a B.P. of 200/110; a trace of albumin in the urine; retinal and radial arteries appreciably thickened; a demonstrable left ventricular enlargement and an accentuated aortic second sound. There is hyperpiesia with arteriosclerosis and cardiac hypertrophy, but there has been no vascular accident or manifest structural damage to any organ. He may have five years to go before some of these late consequences appear. His total prognosis is not likely to exceed ten years."

"In the *third stage* there are symptoms and signs of minor vascular accidents (retinal hæmorrhage, transitory aphasia, or a lesser-grade coronary thrombosis), or of cardiac disability (effort angina, nocturnal dyspnœa). B.P. 220/120. There is hyperpiesia with arteriosclerosis and early structural damage to viscera. The total prognosis is not likely to exceed five years and may be shorter.

"In the *fourth stage* there are symptoms and signs of gross vascular accidents (cerebral thrombosis or hæmorrhage, coronary thrombosis), or of congestive failure. From such accidents clinical recovery cannot be more than partial and temporary. B.P. 240/130. There is hyperpiesia, with arteriosclerosis and serious structural damage to viscera. The prognosis will generally not exceed one or two years."

"In the *fifth stage* the patient is bedridden with chronic heart failure, hemiplegia, or the mental deterioration of a more general cerebral arteriosclerosis. There may for the first time be commencing renal failure. The systolic pressure may have dropped but the diastolic figure remains high, with such readings, perhaps, as 190/140. In the presence of heart failure the readings may be much lower. The prognosis is reduced to days, weeks, or at the most, months. There is hyperpiesia with wide-spread arteriosclerosis and gross permanent damage to one or several organs. The

prognosis of the so-called "malignant" cases corresponds with that of the fourth or fifth stages of the more chronic or gradual form of the disease."

The prognosis of the menopausal cases is much better than that of male cases at the same age, and progress to the stage of arteriosclerotic degeneration is less likely to occur.

The prognosis of malignant hypertension is grave and the fatal end may take place in an average of eight and nine-tenths months as found by Rosenberg.

X. Treatment

Treatment is effective in the first two stages of Ryle's classification, before any manifest structural damage to any organ has been done. Once it has passed the third stage, treatment is almost powerless to affect the duration of life although it may modify disabilities.

Drugs may be useful for secondary symptoms, as in the case of nitrites for anginal pain, morphine for cardiac pain and nocturnal dyspnœa and digitalis for arrhythmia, but for hyperpiesia itself they are of little avail. Ryle says "At no stage should we allow our patient to acquire too keen an interest in his blood pressure readings or report them regularly to him. We should protect him from knowledge of such anxieties as we may ourselves be forced to entertain. As a profession we have erred far too often in the direction of pessimism in hyperpiesia and have attached undue importance to the mercury level as compared with other clinical evidence." He proceeds and says that of all the physical aids, reduction of weight in the obese patient is probably the most important. "A drop in weight and a considerable fall in pressure are sometimes accompanied by a striking symptomatic, albeit temporary improvement." Regarding diet he says "I know no physiological justification for the strict curtailment of red meats, milk and salt, which are often enjoined. In heavy patients, I have thought a reduction of fats and starches more reasonable and more effective." He does not object to open air exercise, but he has often advised rest in bed for one hour a day, one day in a week and one week in each three months to these patients. He agrees that there is no specific treatment for hypertension. Evans and Loughnan[51] exposed by elaborate clinical experiments the futility of almost all the drugs reputed to have hypotensive effect and extensively used.

Regarding surgical treatment, Prof. J. Paterson Ross[52] says "So long as we are satisfied that in spite of the best constitutional and medical treatment, the disease is progressive and that the patient is incapacitated and in pain, the chance of surgical relief should not be withheld." Seventy per cent of his cases derived considerable benefit from the operation of splanchnic neurectomy, their symptoms (particularly the headache) being relieved and their activity restored. The symptomatic relief persisted inspite of a return of the blood pressure almost to pre-operative level, in thirty per cent patients. Twenty per cent derived no benefit. Similarly C.H. Moore[53] reported 22 patients in whom splanchnectomy was done. 90 per cent showed immediate drop of blood pressure. In 45 per cent the blood pressure was maintained for over a year. Symptomatic improvement without fall in blood, pressure was obtained in 27 per cent. In 9 per cent no improvement was noted and 4 per cent died. The results of decapsulation with splanchnectomy and in severer cases, adrenal denervation and decapsulation or removal of the aorticorenal ganglion, were recorded by Chabanier and his associates.[54] The pressure rises in most instances, but the symptom of headache, palpitation and dizziness are relieved.

Omentopexy of kidneys was done in many cases, without any benefit.

The effects of nonspecific operations, such as cholecystectomy, hysterectomy and prostatectomy were studied by Volini and Flaxman[55] in 27 patients. They stated that all patients were relieved of headaches, pain in the chest, dizziness and fatigue regardless of the operation performed. The duration of the symptom-free stage varied from four months to nine years, averaging three and half years. They expressed opinion that the results were better than those of any specific operations. Prof. J. P. Ross says that he did not find any such effects in his patients who underwent hysterectomy and cholecystectomy. He presumes that if spasm of the renal arteries be the cause of essential hypertension, a much simpler operation than splanchnectomy, *viz.*, renal sympathectomy may be as good. We can, however, understand the value of prostatectomy, as it relieves obstruction to urinary flow, causing hypertension.

I have already mentioned Evans and Loughnan's elaborate researches[51] with various well-reputed depressor drugs including some proprietary drugs. 33 drugs were tried on 70 patients, whose age varied from 38 to 72 years and were judged to be cases of hyper-

piesia after routine clinical examination which included urine analysis, tests of renal function, retinoscopy, eletrocardiography, radiocardiography. On radiocardioscopy, left ventricular enlargement and distortion of the aorta in the form of an unfolding was sought. All nephritic cases were excluded. Each patient attended the London Hospital at fortnightly intervals, but whenever a drug required parenteral administration or close observation of its effects, he was admitted into hospital. Questions were asked at each attendance about progress during their trial period, any change in symptoms, the occurrence of any untoward or toxic manifestations, and any variation in the patient's activity or habits which might influence the course of progress. The blood pressure was then taken under conditions which were constant and standard for each patient. Not until their records were completed did they examine the results critically and then it became apparent that although these patients had been taking continuously the many reputed remedies, none showed any clinical change nor any effective change in blood pressure. No evidence was obtained to justify the prescribing of any of the tested preparations, if the object is to reduce the blood pressure, for it was shown that none of them possesses the property under clinical conditions.

The effects of a blood pressure variation were watched particularly where a rise or fall of more than 25 m.m. had taken place. During 64 test periods, when this change was a rise, the symptoms improve in 36 per cent, were unchanged in 38 per cent or were worse in 20 per cent. During 90 test periods, when the change was a fall, the symptoms improved in 34 per cent, were unchanged in 46 per cent and were worse in 20 per cent. The symptoms in a patient with hyperpiesia, therefore, bear no relation to the height of his blood pressure at the time.

The authors expressed surprise to find no improvement in such a high proportion of patients when the pressure had fallen conspicuously and it caused greater surprise to find improvement of symptoms coinciding with a rise as often as with a fall in the blood pressure. Symptomatic improvements were, however, found with bismuth subnitrate, iodine and iodide, bromide, sodium luminal, theominal and potassium thiocyanate. The sedative drugs seem to have value in temporarily relieving nervous symptoms when these were prominent.

It is obvious that a physician should not regard the symptoms as closely related to the height of his blood pressure. Let me repeat the words of Prof. Ryle that undue importance should not be attached to the mercury level as compared with other clinical evidence. We should all agree with the concluding words of Evans and Loughnan when he says that medical attention must be devoted to the alleviation of symptoms when they arise and to correcting the faults in living, and particularly to the adoption of timely active measures, when symptoms or signs of heart failure first appear.

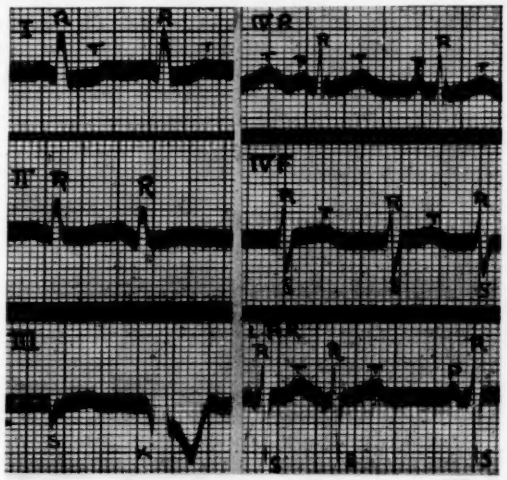
Blood letting, drastic purgatives and such others should never be attempted unless urgent symptoms like hypertensive encephalopathy demand them. Otherwise the pressure gradient of the vessels supplying the renal tubules may fall so low as to interfere with their proper function and ultimately causing uræmia to develop. Injudicious lowering of blood pressure may lead to coronary or cerebral thrombosis, if those vessels were already diseased or sclerosed, as is often the case in hyperpiesia. Prof. Ryle says that the time has come to dispose finally of a number of useless drugs which have been advocated and to discard certain dietetic and other fads which have too long prevailed.

Besides medicinal and surgical treatment, physiotherapy in the shape of short wave radiations were tried in hypertensive cases by Schliephake[56] with excellent results, so far as the symptomatic relief was concerned. He says that there is a preliminary drop of blood pressure in many cases, but it rises to the former level after some time. The relief of symptoms persists in spite of the reversion to the former pressure. U. P. Ganguli,[57] in a recent address in the Calcutta Medical College Reunion, claimed extraordinary results. He cited cases in which application of induced magneto-electric current delayed the undesirable changes in kidneys, especially in malignant hypertension. Failing heart was quickly restored to normal function by induced heat, through dilatation of coronary arterioles and better nourishment of the heart muscle. Schliephake reduced intracranial pressure in hypertensives by application of short waves on the head. This is specially valuable in malignant hypertension. U.P. Ganguli cited cases in which paroxysmal tachycardia, pulsus alternans with gallop rhythm, general anasarca and various other complications of high blood pressure were quickly controlled after exhibition of inductothermy. It opens up a new line of treatment of symptoms and complications of hypertension, both benign and malignant.

Illustrative Case Notes

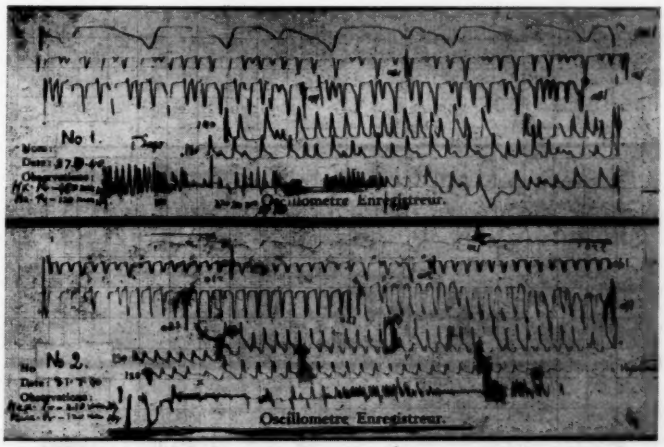
A case of Extreme Left and Right-sided Heart Failure—Case No. 11. S. N. M., Hindu male, aged 60 years. The

patient a well-known politician, and an M. L. A. detected several years ago, that he had high blood pressure, but he



Electrocardiogram No. 1

did not take any particular notice. Since a year, he began to get breathlessness, on heavy exertion. Since then he regulated his activities and habits, and gave up taking meat and eggs. Since October, 1939, he began to lose apetite and gradually all signs of left ventricular failure set in. The bases of the lungs became congested and gradually hypostatic râles appeared. This was quickly followed by right sided failure. The neck veins became prominent, liver was enlarged and gradually œdema, which first appeared in the legs, spread upwards over the whole body. This general ansarca was attended with ascitis and right-sided hydrothorax. The systolic pressure which formerly varied between 230 to 240 mm. Hg. came down to 180 mm. Hg. and the diastolic was reduced from 130 to 120 mm. Hg. The pulse became extremely irregular and frequent extra-systoles made it more so. An oscillograph and an electrocardiogram were taken on 27.6.40. (*See oscillograph No. 1,* and *electrocardiogram No. 1*). The oscillograph showed extreme irregularity of pulse under all pressure and numerous extra-systoles. The blood pressure was 180/120 mm. Hg. The electrocardiogram showed blurring and notching of QRS., indicating extensive degeneration of the myocardium, low voltage of T waves signifying profound myocardial weakness and a left ventricular extra-systole in Lead III, arising high up from near the basal region. The special chest leads (IV R, IV F and L. P. R.) also showed very low voltage of QRS and of T waves. The normal average voltages of R, S and T waves are 20, 7·5 and 7 mm. respectively in Lead IV R, when 1 mv. causes 10 mm. deflection. Here they are 8, 3 and 2 mm. respectively. These indicated myocardial weakness. The patient was advised rest in bed besides digitalis, salyrgan, purgatives and other medical measures, but he persisted in going to the Legislative Assembly with the result that on 20th July, 1939, he was attacked with acute anginal pain in the chest, extreme orthopnœa and oliguria. Leeching, glucose and salyrgan injections failed to give him any relief or increase the quantity of urine which was reduced to four ounces only in 24 hours. He became cyanosed, inspite of continuous oxygen administration. It was at this stage that inductothermy was applied over the precordial area at very low intensity daily for 10 minutes. The patient had not a wink of sleep for three days, but after the inductothermy was applied at 9 p.m., he enjoyed undisturbed sleep for three hours at a stretch, which was gradually increased day after day, until he began to have not only 5 to 6 hours sleep at night but also 2 to 3 hours sleep in day. The quantity of urine was gradually increased to twelve ounces in three days, but when salyrgan was given intravenously on the fourth day of the administration of inductothermy, the quantity of urine passed amounted to fifty ounces in twentyfour hours. This drug failed to secrete more than six ounces of urine previous to administration of inductothermy. The general improvement of the condition started from that time and subsequent administration of salyrgan was attended with copious flow of urine which on one occasion rose to 100 ounces in quantity. The œdema gradually went down, ascitis and hydrothorax disappeared. On 31.7.40 his oscillograph (*See oscillograph No. 2.*) showed steady pulse, and his systolic blood pressure rose to 210 mm. Hg. although his diastolic remained steady at 120 mm. Hg. Since then, the

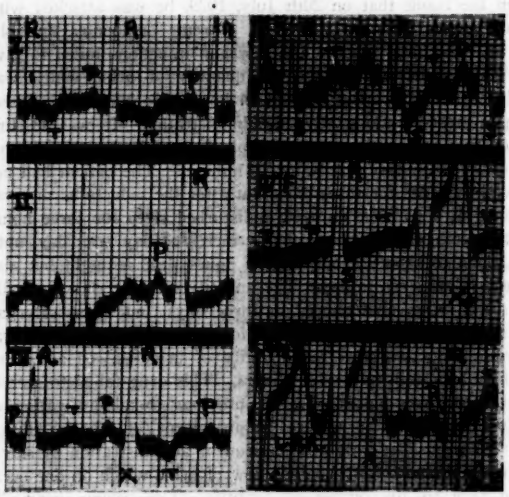


Oscillograph Nos. 1 and 2

patient made steady progress towards increase in exercise tolerance and total disappearance of œdema although the interval between administration of inductothermy was increased to one week.

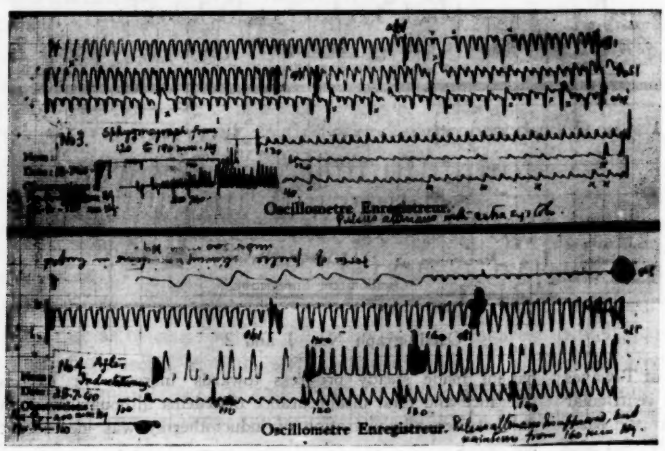
Two Cases of Gallop Rhythm with Pulsus Alternans—Case No. 23. J. C. C., Hindu male, aged 50 years, was seen on 13.7.40 when he complained of profound weakness with a sense of sinking, palpitation, and markedly diminished exercise

tolerance. No cause could be ascertained for the gradually increasing weakness and other symptoms except that he was found to be suffering from high blood pressure. His oscillo-



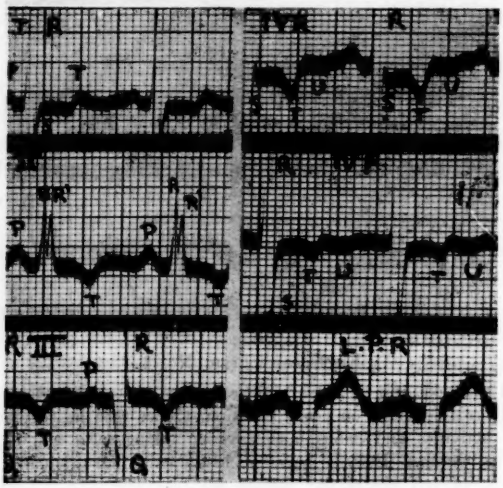
Electrocardiogram No. 2

graph (*See oscillograph No. 3*) showed systolic pressure of 240 mm. Hg. diastolic of 120 mm. Hg., and pulsus alternans in the sphygmographic curves visible under all pressures from systolic to even below the diastolic pressures. There were frequent extra-systoles at places marked by X. The apex was at seventh interspace in the anterior axillary line. Auscultation revealed gallop rhythm. Almost all the authorities in the world are agreed in the opinion that in a combination of gallop



Oscillograph Nos. 3 and 4

rhythm with pulsus alternans the prognosis is grave. It is most commonly seen in patients with high blood pressure when the myocardium is beginning to fail. Its presence means exhaustion of the muscles of the heart, a state with which pathological changes are frequently associated. His electrocardiogram (*See electrocardiogram No. 2*) taken on the same date, *viz.*, 13.7.40. showed both auricular and ventricular extra-systoles in places marked X, inverted T in Lead I, upright T in Lead III, depression or ST segments in Leads I and II, and blurring of tips of QRS complexes. The special chest



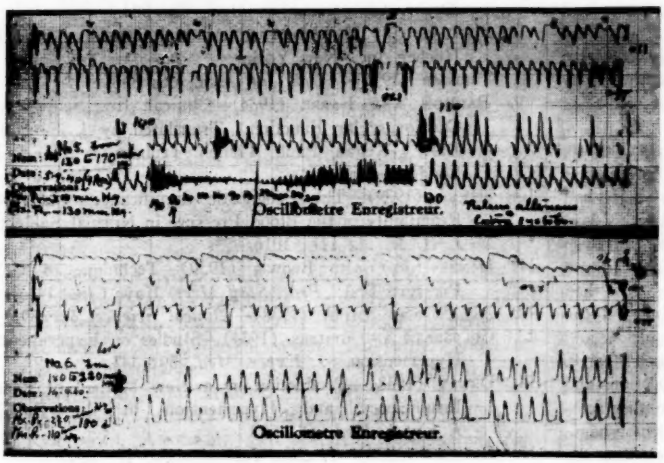
Electrocardiogram No. 3

leads showed still more marked depression of ST segments in Leads IV R and IV F and high "take off" in L. P. R. lead. The myocardium was so much exhausted that after the ventricular extra-systole, the T wave which was ordinarily isoelectric in Lead IV F became actually inverted in the normal systolic complexes following the extra-systoles or became much reduced in voltage, as in Lead L. P. R.

The patient was put to inductothermy, which was begun from 18.7.40 and was applied for 7 days. The subjective symptoms became much diminished and both gallop rhythm and pulsus alternans disappeared. (*See oscillograph No. 4 taken on 28.7.40.*) The patient could not carry on further treatment with inductothermy on account of expense. He was then put on euphyllin, glucose injections and anacardone tablets. Inspite of these measures, pulsus alternans was seen to reappear under systolic and higher pressures on 21st August, 1940, the lower one being free from it, but there was no gallop rhythm.

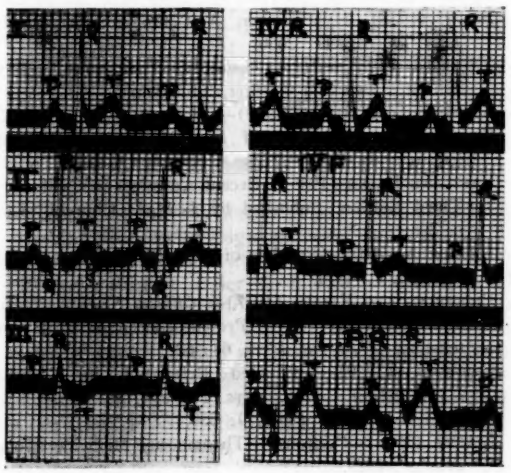
Case No. 189. Khan Bahadur A. S., Mohammedan male, aged 55 years. Patient was seen first on 5.9.40. He complained of breathlessness and substernal pain on slightest exertion. He first noticed these symptoms since a year, but it was

gradually getting worse and since beginning of August 1940, the symptoms became very much pronounced. He had some mental worries and this disturbed him so much, that he felt palpitation, day and night while he was awake. The oscillograph, taken on 5.9.40. (*See oscillograph No. 5*), showed



Oscillograph Nos. 5 and 6

pulsus alternans and high blood pressure of 220/130 mm. Hg. Physical examination revealed enlargement of the left ventricle and gallop rhythm. His electrocardiogram taken a day later, (*See electrocardiogram No. 3*) showed depression of the ST



Electrocardiogram No. 4

segment in Lead I and elevation in Lead III, upright T in Lead I, inverted T in Leads II and III. In the special chest leads (IV R, IV F and L. P. R. leads.) ST segments showed marked depression in IV R and IV F, with inverted T in both. These appearances seem consistent with hypertrophy of the left ventricle according to Wood and Selzer[28] and *relative* coronary insufficiency, on account of the large size of the heart. U waves were also inverted. According to Cornelio Papp[29], "an inverted U wave is the only pathological form yet recognised. It is to be found in coronary thrombosis, hypertension, angina pectoris and aortic incompetence Generally it is found with an inverted T though it can be the only pathological sign in an otherwise normal record." In the present case, it is associated with an inverted T, which is abnormal in both IV R and IV F leads.

Inductothermy was started on 7.9.40. Patient felt improved day by day. In seven days his pulsus alternans disappeared as shown in the oscillograph taken on 14.9.40. (*See oscillograph No. 6*). The extra-systoles were still persisting and it was found that his so-called palpitation was due to heart consciousness about extra-systoles. After another seven days' application of inductothermy, the extra-systoles became few and far between, and his palpitation became correspondingly decreased.

Angina Pectoris in High Blood Pressure—Case No. 5. C. C. B., Hindu male, aged 50 years. The patient's chief complaint was angina of effort. His blood pressure was found to be 175/115 mm. Hg. He was carrying on his professional and political activities, inspite of severe discomfort which he used to feel now and then. Things became very bad in July last, when he had to be confined in bed for nearly a month and euphyllin had to be exhibited. The electrocardiogram (No. 4), which showed high "take off" of T waves, especially marked in special chest leads, indicated marked coronary insufficiency. The descending type of voltage of T waves in standard leads indicated toxic myocarditis in 80 per cent of cases. The patient had history of epidemic dropsy. The patient had to be sent to Ranchi for ensuring perfect rest, as it was found impossible to prevent his patients and political friends from seeking his advice. He got worse, however, after a motor trip, and was brought back to Calcutta in September, when he was given inductothermy treatment. He got marked relief, so much so that after seven applications, he went to Bombay and took part in an all India political gathering of which he was one of the leading and guiding members. He did not feel any pain while carrying on these works in Bombay, but on his return journey, he was attacked with anginal pain in the railway train. Inductothermy was then applied for two weeks, after which the interval was increased to 3 to 4 days and after 23 applications he felt no necessity to continue it any further.

Several other cases of angina, complicated with high blood pressure, were markedly relieved.

Malignant Hypertension with Paroxysmal Tachycardia and Hæmorrhagic Retinitis—Case No. 7. N. B. G., Hindu male, aged 55 years, was suffering from high blood pressure, which was diagnosed as malignant type of essential hypertension. Nine months ago, he had an attack of unconsciousness during which he was found to be suffering from temporary hemiplegia

and amaurosis. After this attack, he suffered from articulate amnesia, which persisted all along, although all other complaints disappeared, except giddiness and excitability on slightest cause. Slightest movement of the head, however, caused the sensation of reeling and he was afraid even to sit up in his bed. Over and above, he began to get attacks of paroxysmal tachycardia. the intervals between each attack getting less and less till it became a daily affair. During these attacks, he used to become restless and slightly dyspnœic. He had also frequent encephalopathic vasomotor disturbances manifested by temporary fits of unconsciousness. Various kinds of medicines were tried for prophylaxis, but to no purpose.

Inductothermy was started from 16th September, 1940, after which the tachycardia ceased to recur. After seven days of daily applications, it was applied on alternate days. He had a slight attack of tachycardia eight days later. A few days later, albumin and casts appeared in the urine and he had fresh retinal hæmorrhages. Inductothermy was then applied on kidneys, alternately with the heart for a few days and then one day's gap was given in between the applications on the kidneys and the heart. The albumin disappeared quickly and after a fortnight's application, the eyeground was much clearer, the old hæmorrhagic patches showed signs of absorption, and there were no further fresh hæmorrhages. After another fortnight, the fundus was free from exudative and hæmorrhagic patches, and only the vessels showed thickening, so that the lumens were contracted. The intervals were increased and inductothermy was completely stopped by the end of December. The attacks of paroxysmal tachycardia and fits of temporary unconsciousness became few and far between, leaving no ostensible after-effects.

N.B.—Cases nos. 11, 23, 189 and 7 have been published in May, 1941 in the Transactions of the Medical College Reunion, 12th Session (1940-41, pp. 167-169) by my assistant Dr. U. Ganguli who carried out the special treatment under my instructions and furnished me with details of the progress of these cases.

It appears that the impending heart failure was averted by exhibition of inductothermy in this case. A study of other cases also proves the remarkable potency of inductothermy or heat-radiation treatment in improving the nourishment of the heart, brain and kidneys, by increasing the blood circulation in those organs through arteriolar dilatation. The amelioration of symptoms is quickly established chiefly through vaso-relaxation. The repair of damaged tissues is also quickly initiated to a remarkable extent.

Jacobson[31] has introduced a system of relaxation of skeletal muscles, which seems to be efficacious in neuromuscular group; the special instructions are contained in the original article.

References

1. Robinson and Brucer (1939)—*Arch. Int. Med.*, 64: 409.
2. Fahr (1935)—*J. A. M. A.*, 105: 1396.
3. Perry and Langsam (1939)—*Arch. Int. Med.*, 64: 971.
4. Halls Dally (1926)—"High Blood Pressure". Published by the Actinic Press Limited, London.
5. Lange (1933)—Arteriosclerose. Published by MacMillan Company, New York; p. 504.
6. Saller (1927)—Blood Pressure Changes Due to Age-*Ztschr. f.d. ges. exper. Med.*, 58: 683.
7. Stieglitz (1935)—Abnormal Arterial Tension. Paul B. Hoeber, Inc., New York.
8. Treloar (1940)—*Arch. Int. Med.*, 66: 848.
9. Pachon and Fabre (1934)—Clinical Investigation of Cardiovascular Function. Translated by J. F. Halls Dally. Kegan Paul, Trench Trubner & Co. Ltd., London.
10. Hines (1939)—The Prognostic Significance of Hyper-Reactibility of the Blood Pressure in Normal Subjects. *J. A. M. A.*, 111: 1016.
11. Hines (Jr.) and Brown (1939)—Technique of Cold Pressure Test. *Proc. Staff Meet. Mayo Clin.*, 14: 185.
12. Allam and Smirk (1938)—*Clinical Science*, 3: 259.
13. Goldblatt and others (1934)—Studies on Experimental Hypertension. *J. Exper. Med.*, 59: 347.
14. Drury (1938)—Production by a New Method of Renal Insufficiency and Hypertension in the Rabbit. *J. Exper. Med.*, 68: 603.
15. Ryland (1938)—*J. Clin. Investigation*, 17: 391.
16. Wilson and Pickering (1938)—*Clinical Science*, 3: 343.
17. Goldblatt and others (1934)—The Production of Malignant Phase of Hypertension. *J. Exper. Med.*, 59: 347.
18. Friedman and others (1939)—*Ann. Int. Med.*, 12: 1617.
19. Hessel (1939)—*Arch. Int. Med.*, 64: 927. ,
20. Prinzmetal and others (1939)—*Ann. Int. Med.*, 12: 1604.
21. Engle and Binger (1939)—*Proc. Staff Meet. Mayo Clin.*
22. Scott (1938)—Hypertension A Century After Bright. *J. A. M. A.*, 111: 2460.
23. Fishberg (1934)—Hypertension and Nephritis. Third Edition. Lea and Febiger, Philadelphia.
24. Schroder and Steel (1939)—*Arch. Int. Med.*, 64: 927.
25. Boyd (1935)—Pathology of Internal Diseases. Second Edition. Published by Henry Kimpton, London. p. 114.
26. East and Baine (1936)—Recent Advances in Cardiology. Third Edition, Published by J. & A. Churchill Ltd., p. 208.
27. Pickering (1937)—Problem of High Blood Pressure man. *Brit. M. J.*, 1: 1.
28. Pickering and Kissin (1937)— *Clinical Science*, 2: 201.
29. Katz and Leiter (1939)—Present conception of Hypertension. *Arch. Int. Med.*, 64: 627.
30. Jacobson (1940)—Cultivated Relaxation in Essential Hypertension. *Arch. Phys. Therapy*, 21: 645.
31. Bell (1928)—*Arch. Path.*, 9: 539.
32. Levine (1929)—"Coronary Thrombosis, Medicine Monographs", 1929.
33. Oppenheimer and Fishberg (1929)—*Arch. Int. Med.* 42: 264.
34. McAlpine (1933)—*Quart. J. Med.*, 2: 463.
35. Ricker (1919)—Virchow's Arch. f. path. Anat., 227: 64. Quoted in Boyd's Pathology of Internal Disease, p. 721.

36. Westphal and Bär (1926)—Deutsches Arch. f. Klin. Med., 151: 1. Referred in Arteriosclerosis by Cowdry, p. 260.
37. Gillan (1939)—Prognostic Significance of Ophthalmoscopic Findings in High Systolic Blood Pressure. *Brit. M. J.*, 1: 609.
38. Rosenberg (1940)—Brain in Malignant Hypertension, *Arch. Int. Med.*, 65: 545.
39. Wagner and Keith (1939)—*Medicine*, 18: 317.
40. Williams and Harrison (1939)—*Ann. Int. Med.*, 12: 1617.
41. Keith, Wagner and Barker (1939)—*Am. J. Med. Sc.* 197: 332.
42. de Wesselo and Thomson (1939)—*Quart. J. Med.*, 81: 361.
43. Page (1939)—*Ann. Int. Med.*, 12: 978.
44. Wilson and Byron (1939)—*Lancet*, 1: 136.
45. Rosenthal and Lander (1939)—*Ann. Int. Med.*, 12: 1449.
46. Davidson and Brile (1939)—Essential Hypertension and Chronic Hypertension Encephalopathy. *Ann. Int. Med.*, 12: 1766.
47. Page (1935)—*Am. J. Med. Sc.*, 190: 9.
48. Ashton Graybiel (1940)—*Arch. Int. Med.*, 65: 1070.
49. Kernohan, Anderson and Keith (1929)—*Arch. Int. Med.*, 44: 395.
50. Ryle (1940)—*Proc. Roy. Soc. Med.*, 33: 342.
51. Evan and Loughnan (1939)—*Brit. Heart J.*, 1: 199.
52. Ross (1940)—*Proc. Roy. Soc. Med.*, 33: 344.
53. Moore (1939)—*Arch. Int. Med.*, 64: 651.
54. Chabanier and associates (1939)—*Ibid.*, 64: 653.
55. Volini and Flaxman (1939)—*Ibid.*, 64: 654.
56. Schliephake (1933)—Short Wave. Third Edition.
57. Ganguli, U. P. (1940-41)—*Transactions of the Medical College Re-Union*, 4: 163.
58. Wood and Selzer (1939)—*Brit. Heart J.*, 1: 49.
59. Papp (1940)—U, the Sixth Wave of Electrocardiography. *Brit. Heart J.*, 2: 9.

Dr. P. KUTUMBIAH'S ADDRESS

Capt. Ganguli opened with an exhaustive review of the present knowledge on the subject. Dr. P. Kutumbiah who spoke next drew attention to the importance of subject under discussion. It is generally stated on very slender evidence that high blood pressure does not exist among Asiatics. Anybody who has worked in a big hospital in South India will be struck with the fact that cases of high blood pressure are very frequent. Among 1200 cases of cardiac trouble treated between 1932-1938 in the King George Hospital, 16 per cent of these were due to hyperpiesis. It is generally regarded that high blood pressure is more common in highly civilised countries as a result of the terrible nervous strain that prevails in those countries. It is not quite true. An analysis of our cases suffering from hyperpiesis shows that we meet with hypertension among people of all walks of life. The intelligentia, the middle classes and also the working classes contribute to the number of hyperpietics treated in our hospital. Though there cannot be any doubt as to the prevalence of hypertension in India, the exact etiology of these cases has not been worked out. A good percentage of these cases are of renal origin and are attributable to chronic parenchymatous nephritis and polycystic kidneys. Cases of suprarenal and pituitary origin are rather rare. There is a good percentage of cases in which no etiological factor has been found. These cases which are usually termed cases of essential hypertension are fairly common in our wards and the etiology of this condition awaits further research. I should like to mention also cases of hypertension which occur during puerperium. These form a very interesting group. The hypertension is amenable to treatment and most of these cases with rest and dieting get completely cured. With regard to etiology, it may be stated that hypertension cases occur as commonly among the vegetarians as among the meat eaters; the teetotalers as well as those who are addicted to alcohol; the smokers as well as non-smokers. From this it is clear that no one factor like excessive protein intake, alcoholism, smoking, can be incriminated as the casual factor. The occurrence of hyperpiesis in post-puerperal cases would point to some temporary mechanism as the probable cause. But this awaits investigation. Three types of cases are met with in our practice. There are a group of cases particularly in women about the time of menopause when the blood pressure is increased and is associated with vasomotor phenomenon. In these cases the blood pressure is very unstable and labile. Occasionally such cases are seen in men also and at an equal age. The other group of cases are those in which there is high blood pressure with no marked cardiovascular hypertrophy. In these cases who are generally males and about thirties, the blood pressure is fairly fixed and the last group and the most important group of cases are those in whom there is permanent fixation of the blood pressure with cardiovascular hypertrophy later followed by retinal changes, etc. I would specially impress these three stages of the disease. The first of these groups are from the point of view of prophylaxis and treatment, the most amenable to treatment. Attention should be paid to these cases with a view to discover what it is that causes high blood pressure. One other fact with regard to the hyperpietic patient is that they do not come for treatment until some major catastrophe occurs—either a cerebrovascular accident or cardiac decompensation or uræmia. When the heart is compensated these people are robust in health and have a great deal of energy and capable of good exercise. This is why the prognosis of these cases of high blood pressure which we see in the wards is so grave. With regard to treatment I would suggest that it is (1) inadvisable to unnecessarily alarm the patient about his pressure; (2) no unnecessary and drastic interference with the regimen of life followed by the patient to be attempted; (3) hypertensile drugs should not be used or if used at all should be used with a great deal of caution. It is a matter of common experience

that our intelligent patients react psychologically very badly to a knowledge of having blood pressure. It does no good for the patient to be constantly worried about his blood pressure. As we know definitely in most cases it is not the blood pressure that is of any great prognostic significance but it is the presence of symptoms. So it is advisable not to unduly alarm the patient about his blood pressure. It may be explained to him that as far as the blood pressure is concerned nothing could be radically done to lower it and even if it could be lowered it is not good for him, since he has got accustomed to a certain level of pressure. With regard to the intake of diet it has not been proved conclusively that excessive proteins or alcohol or tobacco has any definite etiological relationship with high blood pressure. But it is absolutely essential that any regimen of life should be one which will have the confidence and co-operation of the patient and you should not impress upon the patient that he is an invalid and is on the verge of disintegration. I would suggest that moderation in all things, food, drink, smoking, sexual relationships, exercise, as the most useful formula for the regimen of life. It should not be understood that either alcohol or tobacco is advocated but where a patient is addicted to these, he should be advised to cut it down to the lowest possible minimum. The question of exercise is one of great difficulty. It entirely depends on the integrity and the reserve power of the cardiac muscle; as long as the patient carries on his usual work with no evidence of cardiac distress like precordial or retrosternal pain, dyspnœa, throbbing of the head or dizziness, there is nothing to interfere with that amount of exercise. When the blood pressure shows a tendency to fall after any exercise it should be given up. With regard to diet I have found of late a regimen of semi-starvation very beneficial. In the night, fruits and a cup of milk, a square meal in the afternoon, will be found to be very suitable by most of the patients. I would specially mention the importance of pulse rate in the treatment. High blood pressure before decompensation occurs, is often associated with bradycardia, which is nature's method of conserving the reserve powers of the heart and resting of the heart. This should be imitated in our treatment also. Whenever the pulse rate is above normal it should be brought back to the normal level either by complete rest or by the administration of digitalis. Tincture digitalis is a drug of immense importance in the treatment of hyperpietic cases and should be given more confidently and liberally when there is any evidence of cardiac decompensation, than the orthodox school will allow. However, with regard to the use of hypotensile substances in treatment, if our present knowledge of the pathology of the disease is correct, it is very questionable whether any hypotensile drug will be of any use once the blood pressure is fixed, and cardiovascular hypertrophy has occurred. And it is at this stage we are called upon to treat most of our cases. Amyl nitrite is certainly our main standby with acute cardiac distress threatening and is used symptomatically to relieve this. Its use is purely of temporary value. Cases in which blood pressure is not fixed do certainly respond well when nitrites are exhibited occasionally. I have seen cases in which one grain of sodium nitrite has controlled blood pressure satisfactorily for over a period of one week. But the advanced cases do not respond to nitrite therapy, in whatever form it is administered, and even if they do respond the question whether it is desirable to lower the blood pressure arises. I am firmly of opinion that reduction of blood pressure is attended with grave dangers in cases of the third group. It might lead to cerebral thrombosis. I am absolutely convinced that no attempt should be made to lower the blood pressure by hypotensile substances. But any sudden rise of blood pressure should be controlled by complete physical and mental rest. Venesection as such has no effect on the blood pressure, as I have measured the blood pressure when venesection was being done and about 20 ounces of blood was taken the pressure dropped only about 5 mm. of mercury. But where there is evidence of congestion, venesection will be lifesaving. Another important drug in the treatment of hypertension is bromide. On account of its sedative action on the cerebral centres, its use is to be advocated as a routine in all cases of hyperpiesia. Whenever the patient complains of insomnia or restelssness in the night he should be given bromide and chloral hydras mixture every night till it is controlled. The other drugs which on theoretical grounds are to be advocated are dilators of the coronary arteries. It is questionable whether the arteries of a hyperpietic patient respond to any drug producing vasodilatation. Anyway the systemic arteries do not show much evidence of vasodilatation under the administration of nitrites. Drugs like theobromine may be tried.

There are many problems connected with high blood pressure which await thorough discussion by the conference. I have just hinted on few practical things which have struck me during the course of my limited experience. The subject is of very practical importance and is well worth a discussion by those who are assembled.

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HEART FAILURE IN HIGH BLOOD PRESSURE

In the opening address of a symposium on high blood pressure at Vizagapatam, published in this issue of the Journal, Ganguli stressed on the fact observed by Cabot in his book on Heart and Disease that hypertensive heart disease is more common than all other forms of heart diseases combined. This fact was also corroborated by Perry and Langsam,[1] in their post mortem findings of 2,066 consecutive autopsies from cardiovascular diseases, 55·8 per cent of which died of hypertensive heart disease. One of the effects of essential hypertension is the gradual involvement of the heart, which comes on sooner or later in the shape of enlargement of the left ventricle. This enlargement is to be regarded as an indication of a diseased heart for it is worth while to remember the dictum of Campbell that *while all diseased hearts are not enlarged, yet all unquestionably enlarged hearts are diseased.* So long as the patient keeps his activities restricted up to the limit of endurance of the overloaded heart, the size of a healthy heart remains unchanged inspite of high blood pressure. But if the patient acts indiscreetly and if the heart is diseased or its blood supply insufficient to maintain its nourishment, the left ventricle yields and begins to enlarge. Sometimes dramatic symptoms may develop in the shape of sudden breathlessness at night, known as cardiac asthma, during which both systolic and diastolic pressures are increased. The prognosis of the cases in which cardiac asthma supervenes is to be regarded as very grave.

Fortunately very few cases of high blood pressure develop this dangerous complication. The progress of the disease is very slow except in malignant variety of essential hypertension in which the kidneys mostly bear the brunt of the disease. In ordinary cases, so long as the left ventricle bears the load of high pressure, and even hypertrophies as a reaction, the blood pressure is sustained or increased but a sudden drop of the systolic blood pressure without corresponding decrease of the diastolic one is almost a sure indication of dilatation of the left ventricle. This fall of blood pressure should be regarded as an evil omen, for it is a precursor of right ventricular failure. The first effect of left ventricular failure is, however, an increase of stress on the left auricle. It is exceedingly important to determine when this stress begins. The sudden fall in systolic blood pressure occurs as a result of gross dilatation of the left ventricle but the beginning of the stress on the left auricle can be determined much earlier by electrocardiographic changes of the P-waves. Paul Wood and A. Selzer[2], have found a new sign of left ventricular failure in cases of hypertensive heart disease in which a widened P-wave of à low voltage, usually bifid or flat-topped is found and they suggest that such P-wave resulted from left auricular stress.

The next picture is the slowing of the rate of circulation in the lungs. The most important clinical signs are increase of intensity of the pulmonary second sound and commencing dyspnœa on exertion, whose intensity subsequently runs parallel to the degree of stasis of the pulmonary circulation. The measurement of vital capacity of the lungs gives us a clue to the degree of pulmonary stasis in heart failure. As the stasis increases, dyspnœa becomes more and more evident and the advanced picture of pulmonary stasis is orthopnœa along with transudation of fluid into the alveolar processes.

When pulmonary stasis sets in, there is stress on the right heart, which gradually leads to its dilatation hyperpietic cases. The visible manifestation of right-ventricular failure has a bad prognostic significance in hyperpietic cases. The visible manifestation of right sided failure is the gradual appearance of engorged vein in the neck, the height and extent of which gives us a measure of the failure. Pulsation of these engorged veins increases as the tricuspid valves become incompetent to check the backward flow of the blood during systole. According to Sir Thomas Lewis, the increase of venous pressure can be accurately gauged from the vertical height of the venous engorgement and pulsation from the first creeze in the neck above the level of the manubrium sterni with the patient lying down. It is important to remember that rightsided failure brings on some relief on pulmonary stasis and temporary

[1] Perry and Langsam (1939)—Arch. Int. Med., 64: 971.

[2] Wood and Selzer (1939)—*Brit. Heart* J., 1: 81.

relief of dyspnœa, but as the venous engorgement of the neck is increased, urinary secretion is decreased. The filtration of urine is inversely proportional to the venous pressure. Judicious bleeding by withdrawal of blood from a vein or leeching may, at this stage, bring on a temporary relief to the right ventricular musculature, causing increase in the urinary secretion. This is specially important when the reserved spaces of the liver become more than engorged with venous blood. The next downward step comes on in the shape of capillary stasis or general circulatory failure leading to œdema of tissues, interfering with metabolic activity of all the tissue cells of the body.

Once the right ventricular failure supervenes on the top of the left ventricular one in cases with high blood pressure, the closing of the scene becomes only a matter of time. It is, therefore, extremely important to guard against the onset of the right ventricular failure, the signal of which is given by electrocardiographic changes of P-waves indicating stress on the left auricle.

AETIOLOGY OF HEART DISEASES IN INDIA

Rheumatic heart disease is mainly a disease of the colder and wetter parts of the temperate zone. The disease is also prevalent in the colder and wetter seasons of the year *e.g.*, autumn and winter in the United Kingdom. Western authors have repeatedly pointed out that the disease is unknown in the tropics. Emphasis has been laid on the warm climate more than anything else. Megaw[1] pointed out that the disease in the tropics was contracted more in the hills than in the plains. Rogers[2] reported only one case out of 4,800 post mortem examinations. Since then Basu[3 4 5], Hughes and Yusuf[6], Stott[7 8 9], Hodge[10 11 12], Carruthers,[13] Banerjee,[14] Kelly[15] and Kutumbiah[16 17], have recorded the existence of rheumatic heart disease in the plains of Bengal, United Provinces, Punjab, Madras and Bombay Presidencies. Gunewardene[18] in Ceylon found 20 per cent in hospital and 8 per cent in private practice of rheumatic heart disease in all cardiac cases. Raghavan, whose paper is published elsewhere, has reported 6·1 per cent in 568 heart cases out of 4335 autopsies done in the K. E. M. Hospital, Bombay. We think it has been proved very decisively that rheumatic heart disease is prevalent in the tropics but the incidence is much lower than in England (51 per cent) or America (New England 39·5 per cent)[19]. It looks that the question of influence of climate on the ætiology of rheumatic heart disease should be more thoroughly investigated.

Syphilis is not an uncommon malady in this country. Very little work has been done on its cardio-vascular complications. Raghavan has shown that the incidence of cardiovascular syphilis is 3·9 per cent, which compares favourably with the figures of New York, Boston and Glasgow. Gunewardene's[18] figures are 10 per cent in hospital and 5 per cent in private practice. Most of the hospital class of patients in this country go without treatment for syphilis, as in most of the hospitals these patients are to pay for the drugs which they cannot afford. On the contrary, majority of cases in the West get thorough treatment in the hospitals and venereal clinics. Inspite of this contrast in the treatment, the figure of Raghavan is almost equal to that of the Western World. Does it mean then that the treatment of syphilis does not affect the cardiovascular complications in later life? It must be admitted that adequate treatment for *early* syphilis certainly protects the majority of patients so treated against subsequent cardiovascular syphilis. In 1914 Cabot reported 12 per cent of a group of 6,00 cardiac cases as due to syphilis and in 1928 White and Jones[19] reported only 5 per cent. This is attributed to early diagnosis and treatment of syphilis.

[1] Magaw (1910)—*Indian M. Gaz.*, 45: 81.
[2] Rogers (1925)—*Glasgow M. J.*, 103: 95.
[3] Basu (1925)—*Indian M. Gaz.*, 60: 307.
[4] *Idem* (1932)—*Ibid*, 67: 566.
[5] *Idem* (1941)—*Ibid*, 76: 11.
[6] Hughes and Yusuf (1930)—*Indian J. M. Research*, 18: 483.
[7] Stott (1930)—*Indian M. Gaz.*, 65: 65.
[8] *Idem* (1938)—*Ibid*, 73: 271.
[9] *Idem* (1939)—*Bull. Office internat. d'hyg. pub.*, 26: 623.
[10] Hodge (1932)—*Indian M. Gaz.*, 62: 241.
[11] *Idem* (1935)—*Medical College Magazine, Calcutta*, 9: 1.
[12] *Idem* (1938)—*Indian M. Gaz.*, 73: 275.
[13] Carruthers (1936)—*Ibid*, 71: 137.
[14] Banerjee (1935)—*Indian J. Pediat.*, 2: 279.
[15] Kelly (1940)—*Indian M. Gaz.*, 75: 129.
[16] Kutumbiah (1935)—*Indian J. Pediat.*, 2: 631.
[17] *Idem* (1940)—*Indian J. M. Reserach*, 22: 631.
[18] Gunewardene (1935)—Heart Diseases in the Tropics. Butterworth & Co. Ltd., Calcutta.
[19] White (1937)—Heart Disease, Macmillan Co., New York.

The incidence of hypertension in the series of Raghavan is 11·9 per cent of the total. Gunewardene[18] found 13 per cent in hospital and 25 per cent in private cases. Sen Gupta[20] in an analysis of a series of 838 cardiac cases between 1931-1935 has shown that hypertension is on the increase (7·8 per cent in 1931 to 18·3 per cent in 1935). Modern method of living with its anxieties and worries has much to do in the increase of hypertensive cases. Hypertension, on the other hand, is one of the important ætiological factors of coronary thrombosis. Raghavan's paper does not show the incidence of this grave and interesting malady. Sen Gupta[20] in the same series of 838 cases, found coronary thrombosis only in 18 cases (2·1 per cent). From Indian medical literature he could find only 8 cases reported by Sen Gupta[21], Ganguli[22], and Gupta[23]. It is unfortunate that proper attention has not been given to diagnose this very important clinical entity. Electrocardiographic examination during life and careful post mortem examination of the branches of the coronary artery and of the cardiac muscle and post mortem rœntgenogram of the heart after injection of opaque substance in the coronary vessels or examination of serial section should be frequently done in order to ascertain the incidence of coronary thrombosis.

On the whole the investigations carried in cardiovascular disease are still unsatisfactory. We only possess scattered and largely incomplete data of small or limited scope from a number of sources. Sources like life insurance statistics, periodic health, school, industrial, athletic and military examinations, hospital and mortality figures must be thoroughly taken advantage of. There should be co-operation between private practitioners and hospital authorities in any investigation. In this country the growing menace from cardiac diseases in all decades of life with their crippling effect is not perhaps fully realised.

[20] Sen Gupta (1940)—*Calcutta M. J.*, 37: 359.
[21] Sen Gupta (1935)—*Med. College Mag., Calcutta*, Vol. IX.
[22] Ganguli (1935)—*Calcutta M. J.*, 30: 129.
[23] Gupta (1936)—*Calcutta M. J.*, 30: 471.

GOVERNMENT RECOGNITION TO BIHAR PROVINCIAL BRANCH, I.M.A.

We are glad to announce that the Government of Bihar have decided to include the Bihar Provincial Branch of the Indian Medical Association in the list of Associations recognised by the Bihar Government. This is a very welcome news and we congratulate the Government of Bihar on their wise decision and we can assure them that the Provincial Branch of the Indian Medical Association in Bihar will render all assistance to the Bihar Government in the matter of technical and expert advice regarding all public health measures and problems, on which advice may be sought from the Branch. It may be noted in this connection that the Government of the United Provinces was the first to accord recognition to the Provincial Branch of the Association in U.P. and the Government of Bengal have also recently accorded official recognition to the Bengal Provincial Branch of the I.M.A. These two Governments have also been consulting and taking the advice of the Provincial Branches in all matters affecting public health policy within their jurisdiction. This practice, it may be mentioned, is also availed of in Great Britain and the Dominions, where the British Medical Association and its branches are frequently consulted by the Governments in matters affecting medicine, public health and medical education.

We hope the Governments of the other Provinces in India, who have not yet recognised the Provincial Branches of the I.M.A. in their provinces, would similarly accord recognition to these branches as early as possible.

WANTED A LIBRARY

We are feeling every day the necessity of a good reference library. The Journal of the Indian Medical Association is suffering from this want all along. It is high time that steps should be taken by all concerned to remove this long felt and everyday growing want.

The Editor, Journal of the Indian Medical Association, will gratefully accept any book, journal or medical publication, recent or old, which members may kindly present. Such gifts will be duly acknowledged in the columns of the Journal. Gifts of monographs and books written by members will be specially appreciated.

NILRATAN SIRCAR,
Editor, J.I.M.A.

CURRENT MEDICAL LITERATURE

MEDICINE

Premonitory Symptoms of Acute Coronary Occlusion: A Study of 200 Cases

Master and Others (*Ann. Int. Med.*, 1941:14:1155) from the analysis of detailed histories of 260 patients with acute coronary occlusion report that premonitory symptoms were present in 44·2 per cent cases and they consisted of substeranal or precordial pain or discomfort in most cases and fatigue, weakness, gastric distress, dyspnœa, palpitation, nervousness and dizziness in others. They further note that these symptoms usually appeared within 24 hours prior to the acute attack but in some cases they began two or three weeks before, that they lasted for from a few minutes to several hours, that the onset of the symptoms occurred during rest in 28·5 per cent, during mild or moderate activity in 68·5 per cent and during strenuous effort in 2·9 per cent, that these premonitory symptoms were not associated with clinical evidence of myocardial infarction as evidenced by the absence of fever, leucocytosis, tachycardia, drop in the blood pressure and characteristic electrocardiographic changes (these factors are significant in differential diagnosis). The authors assume the anatomic basis for the premonitory symptoms to be a gradual occlusion of the lumen of the coronary artery by progressive or recurrent intramural hæmorrhage or by primary thrombosis on a plaque which many take hours or days for completion. They maintain that the early recognition of the premonitory symptoms should lead to a reduction of heart failure and decrease the mortality rate by means of immediate bed rest, which, however, will probably not prevent the impending occlusion.

Effect of Intravenous Solutions On Patients With Or Without Cardiovascular Defects

Murphy and Others (*J.A.M.A.*, 1941:116:104) observe that no tests of cardiovascular function will enable one to determine before hand that the patient will respond unfavourably to fluids administered intravenously and careful clinical examination to determine the presence and absence of heart disease is still the best preventive for unfavourable reactions to fluid. In presence of heart disease, regardless of the state of compensation, fluids must be given slowly, in small volumes, preferably isotonic and repeated at intervals of not less than four to six hours. The danger of fluid injection in cardiac patients may not be in increasing blood volume *per se* but rather in farther altering the already disturbed chemistry of the body fluid, thus increasing osmotic pressure derangement. The ultimate fate and mode of distribution or loss of injected fluid is dependent on the kind and concentration of the solution, state of hydration of the patient, degree of cardiac compensation, level of venous pressure and chemistry of body tissues influencing osmotic pressure. Physiologic solution of sodium chloride may prove more useful in increasing blood volume than hypertonic solutions. Non-cardiac patients in the older age groups tolerated fluids as substitution therapy in amounts up to atleast 3,000 c.c. daily in 1,000 c.c. doses at rates of 20 to 40 c.c. per minute, even in absence of dehydration. Alternation of dextrose with physiologic solution of sodium chloride obviated the changes in blood dilutions, weight gain and occult and vissible œdema occuring with physiological solution of sodium chloride alone. The indiscriminate use of intravenous fluids, specially for persons with cardiovascular defect, should be discouraged and safeguards suggested be more strictly adhered to.

Treatment of Subacute Bacterial Endocarditis: Present Status

Lightman and Bierman (*J.A.M.A.*, 1941:116:286) observe that among 200 cases of subacute bacterial endocarditis due to streptococcus viridans and non-hæmolyticus collected from the literature and the records of the Mount Sinai Hospital in which the sulphanilamide drugs were administered, recovery occurred in 12, an incidence of 6·0 per cent recovery. Among 43 patients treated with combined chemotherapy and heparin 5 recovered, an incidence of 11·65 per cent recovery. Among 24 patients treated by chemotheraphy and physically induced hyperthermia 4 recovered, an incidence of 16·0 per cent recovery. Of 21 patients treated by chemotherapy and hyperthermia induced by intravenous typhoid-paratyphoid vaccine 5 recovered, an incidence of 25 per cent recovery. Though the number of cases treated by the combined therapy is very small the results obtained are encouraging and suggest further trial of the method.

Treatment of Staphylococcal Septicaemia with Sulphamethylthiazol and Sulphathiazol: A Report of 12 Cases

Hamburger and Riegsegger (*Ann. Int. Med.*, 1941:14:1137) report 12 consecutive cases of staphylococcal septicæmia treated with thiazol derivatives of sulphanilamide with recovery in 8 cases and that in all the fatal cases endocarditis was present at autopsy. A skin rash and an instance of peripheral neuritis were probably attributable to sulphamethylthiazol. Conjunctivitis and chills and fevers were observed as examples of sulphathiazol toxicity. It is also noted that between the years 1933-39, 27 cases of staphylococcal septicæmia were recorded with only 4 recoveries.

Adrenal Cortex Extract in the Treatment of Bromide Eruption and Bromide Intoxication

Bondurant and Campbell (*J.A.M.A.*, 1941:116:100) write that adrenal cortex when given in combination with sodium chloride is apparently of value is the excretion of bromide and bromide effects (adrenal cortex restores the normal physiologic level of chloride). They further note that sodium bicarbonate is of value in controlling the œdema seen in extensive chloride therapy.

Diagnosis and Treatment of Gouty Arthritis

Hench (*J.A.M.A.*, 1941:116:453) mentions the following as characteristics of gout, *viz.*, (1) arthritis, (2) abnormality in the renal excretion of uric acid, (3) hyperuricæmia, (4) deposits of sodium urate crystals in articular, periarticular, subcutaneous tissues and in kidneys and (5) frequent terminal vascular lesions in the kidneys, heart and brain. The ætiology

is unknown but the factors responsible for the disease are (a) some endocrine deficiency, (b) abnormal renal insensitivities to urates, (c) selective renal insufficiency for urates alone, (d) a distrubance of the vegetative nervous system, (e) allergic reaction to proteins, to purine as well as nonpurine foods (endogenous and exogenous origin). Symptoms are quite characteristic. Diagnosis is easy in advanced cases with tophi, hyperuricæmia and podagra but in early cases the diagnosis is based on certain features no one of which is diagnostic but a combination of which is almost pathognomic for gout. Gouty attacks occur soon after some trauma, or dietary indiscretion or infection, involve most often peripheral joints of feet, ankles, hands and wrists; the affected joint is often warm and red or bluish, the skin over it is shining with some œdema and desquamation later on. Pain is marked but recovery is rapid. *Treatment of an acute attack.* Prevention in children and relatives of gouty patients (showing symptomless hyperuricæmia) by avoiding dietary excesses and other provocative causes of gout. Curative—In actual attack the opening of the bowels by salines, rest in bed, local application of heat or cold and the use of colchicine (which is more effective than vinum colchicum) and narcotics (a dose of codeine or morphine sulph may be necessary until the pain is controlled by colchicine) are necessary. Cincophen and its substitutes (in 7½ grains doses) may also be of value. Diet should be free of purines, low in fat, rich in canbohydrates and purine free proteins and there must be complete abstinence from alcohol.

Interval treatment of symptomless gout and control of hyperuricæmia. Diet should be of low purine content. Coffee and tea are permissible but alcohol not. Patients allergic to certain food stuffs should avoid the particular food. Urate elimination—actylsalicylic acid, sodium salicylate, sodium salicylate 'augmented' by amino-acids, neocinchophen or cinchophen are some of the urate eliminants. Cinchophen and neocinchophen though very effective are toxic drugs and should be used cautiously. 7½ grains are given thrice daily for 3 days a week. 2 litres of fluid daily and liberal amounts of carbohydrates are necessary to avoid toxic effects of the drug (anorexia, dyspepsia, nausea, pruritus and jaundice). Alkalies —potassium citrate from 15-30 grains thrice daily should be taken during the period of using urate eliminants to prevent renal colic from the precipitation of urate gravels in acid urine. *Treatment of chronic gouty arthritis.* Prevention by the methods mentioned above is necessary. When actually present physical therapy as heat, massage, exercise and spa treatment may be used cautiously.

Treatment of tophaceous ulcers consists of excision and removal of the tophii.

Treatment of renal and other complications. The treatment of gouty nephritis involves a generous intake of fluid (2—3 litres), restriction of protein in the diet and continued efforts to combat the underlying gout.

Prognosis is best for those in whom symptoms develop late, poorest for those in whom symptoms develop early in life, specially in the teens. The treatment must be persistent and prolonged. The infective and metabolic diseases associated with it must also receive attention.

SURGERY

Current Trends in Diagnosis of Renal Tuberculosis

Emmett (*Bull. New York Acad. Med.*, 1941:17:45) from an analysis of the methods of diagnosis employed in 216 cases of renal tuberculosis encountered at the Mayo Clinic in the past five years points out that the trend of diagnosis is away from retrograde pyelography and toward excretory urography. An appraisal of the various methods of diagnosis employed in these cases suggests that in the majority of the cases accurate diagnosis can be made by means of excretory urography and one cystoscopic examination for the purpose of obtaining specimens of urine from the good kidney and at times both kidneys to be examined microscopically as a wet smear, with the acid fast stain and by inoculation into guineapigs. It is seldom necessary to perform more than one cystoscopic examination on each patient and multiple cystoscopic examinations are to be avoided. Retrograde pyelograms of the good kidney may be indicated when visualization by excretory urography is not adequate. In the average case retrograde pyelograms of the 'bad' kidney are not necessary even though visualization by excretory urography is incomplete or absent. There is, however, a group of cases in which the diagnosis of renal tuberculosis is difficult and in these bilateral retrograde pyelograms must be employed. The routine use of the latter is to be condemned as an unnecessary diagnostic procedure which is not in the interest of the patient.

The Therapeutic Significance of Plasma Protein in Severe Burns

Elman (*J.A.M.A.*, 1941:116:213) point out that the general manifestations of a severe burn develop rapidly and if they are combated quickly many lives will be saved. These manifestations are due largely to rapid and extensive loss of both tissue and specially plasma proteins. Replacement of the latter by plasma transfusion is essential to maintain the circulation and to relieve the concentration of the blood. A high protein diet is also necessary as soon as nourishment by mouth is possible. A single injection of acacia may be used if no blood is available but it is greatly inferior to plasma. The injection of large amounts of dextrose and saline solution alone in severe burns may prove not only ineffective but may even produce deleterious results.

Adrenal Cortical Hormone in Surgery

Corrado (*Med. Times*, 1941:69:155) in reporting on the effects of adrenal cortical hormone on patients prepared for major surgical operations by giving orally 3 tablets daily for periods of from 10 to 30 days prior to operation observes that these hormone-treated patients withstood the stresses of anæsthesia and operations much better than the patients not so treated—the blood presure, pulse rate and plasma volume as measured by red call counts and per cent of hæmoglobin showed much less fluctuation than in the 'controls'. The shock symptoms were absent or very slight in the hormone-prepared patients. The author holds the view that adrenal cortical hormone very probably effects it beneficial action by maintaining a better regulation of the fluid and electrolyte balance of the

blood and tissues with little change from the normal in cell membrane permeability. The author stresses on the utility of the preoperative prophylaxis with the hormone rather than attempted heroic treatment with large amounts of injectable extract after shock is established.

Medical Management of Fractures

Nelson and Nelson (*J.A.M.A.*, 1941:116:184) write that the object of this management is to establish a serum calcium concentration of from 10·5 to 12 mg. per 100 c.c. and a serum phosphorus concentration of from 3·5 to 4 mg. per 100 c.c. with a ratio of 3 calcium to 1 phosphorus. In addition to milk and calcium medication the authors suggest the use of parathyroid to assist calcium metabolism, of vitamin D to assist phosphorus metabolism and dilute hydrochloric acid to ensure the absorption of calcium and phosphorus from the intestines. The determination of serum calcium and phosphrous will be of value in instituting the treatment but where there is no facility for blood chemistry the treatment mentioned above may be tried without it as at least 85 per cent of all cases of fracture present a low serum calcium.

OBSTETRICS AND GYNAECOLOGY

Recent Advances in Gynaecologic Endocrinology

Greenhill (*Illionis M.J.*, 1941:79:61) writes that three new preparations have been introduced into gynæcologic therapy during the past two years and these are (1) the male hormone, testosterone propionate, (2) the gonadotropic hormone derived from pregnant mare's serum and (3) stilbestrol. The first two are hormones but stilbestrol is a chemically-prepared product. Testosterone proprionate is useful in some cases of excessive uterine bleeding, dysmenorrhœa, painful breasts, premenstrual tension and menopausal disturbance. This hormone should be used with caution in women because of the possibility of producing signs of virilism. The gonadotropic hormone is indicated in women who do not ovulate. Endometrial biopsy revealing abscence of ovulation may be necessary before using this hormone. Stilbestrol has got numerous properties of the œstrogens and may be used to relieve menopausal disturbances, gonorrhœal valvovaginitis, senile vaginitis and pruritus vulvæ. It is cheap and highly effective when given by mouth but its toxic effects must be considered before it is used freely. In addition to the customary routes of administering hormones, namely by the hypodermic route, by mouth, through the vagina and rectum, hormones are now being applied through the skin and under the skin. The subcutaneous inplantation of pellets of hormone preparations or "artificial ovaries" will prove most useful for women who will presumably have distressing menopausal symptoms over a long period of time. The author lays great stress on the caution to be excercised in using new endocrine preparations.

Treatment of Toxaemia of Pregnancy

Murray (*Practitioner*, 1941:146:1) deals with the subject under the following heads:

Hyperemesis gravidarum—This may be of nervous and toxic types. The nervous type may be present without any toxic signs or symptoms but the resulting starvation acidosis may give rise to a state of profound toxæmia. Treatment—Bowels must be opened with calomel grains three followed by saline. Water, barley water, fruit juice or glucose water should be given until bowels are functioning well. Fluids and glucose are given by mouth or if necessary by the rectal route or intravenously. Diet is increased slowly and cautiously. Persisting vomiting, the presence of albumen or bile in the urine, icterus, progressive emaciation and a persistingly high or rising pulse rate are disquieting features. Termination of pregnancy should be done if the pulse rate is persistently over 120 inspite of treatment and if there are other toxic symptoms. The presence of jaundice is a clear indication and so is the occurrence of hyperemesis late in pregnancy.

Albuminuria—It is rarely present before the 7th month but if present before the mid-term suggests underlying kidney trouble. Signs and symptoms present are: œdema, albumen in the urine, raised blood pressure, vomiting and loss of appetite. The condition should always be considered as toxic and so treated. Treatment—Rest in bed, fluids and opening up of the bowels; restriction of proteins in the diet and careful limitation of work which probably will maintain the balance and allow the pregnancy to go to term. Increasing amount of albumen in the urine, markedly raised blood pressure such as 160 mm Hg. or more, recurring œdema, vomiting and headache or eye symptoms are some of the indications for termination of pregnancy.

Eclampsia. It is merely a severe case of albuminuria with fits. The presence of epigastric pain or twitchings are of special import. Treatment. Injection of morphine gr. ¼ when fits are imminent or actual and repeated if they occur after 30 minutes following the injection. Chloral and bromide 20 grains each may be added or given later. Enemas or better colonic lavage may be used when the patient is unconscious and aperients cannot be given by mouth. The room should be quiet, warm and darkened. Atropine sulph gr. 1/60 may be given if there is pulmonary œdema and the patient should not be allowed to lie on one or other side for longer than 2 hours at a time. Warmth is applied to promote perspiration. Venesection should only be done if there is high blood pressure and 6-10 ounces of blood may be removed from the median basilic vein. Intravenous salines are contraindicated as they increase the risk of pulmonary œdema. If labour does not start the question of termination of pregnancy may be decided. All cases of toxæmia should be kept on a protein free diet for 48 hours after delivery and given aperient to ensure an early movement of the bowels.

Acute yellow atrophy. The prognosis is bad and the condition is usually fatal. The only chance of saving the patient is the termination of the pregnancy.

Termination of pregnancy is done in the later months of pregnancy by rupture of the membranes by means of a metal female catheter, in the early months by means of the blunt curette or ovum forceps and in the intervening stage *i.e.* about the 3rd and 4th months by a vaginal or abdominal hysterotomy if it is done in one stage and by a dilatation and cervical plugging if done in two stages. The anasthetic used may be general or local.

EMERGENCY COMMISSION IN THE I.M.S.

The following are press reports of the speech of Major General R. H. Candy, I.M.S., Surgeon-General with the Government of Bombay, at the meeting of the Bombay Branch of the British Medical Association, held on the 16th June 1941, as published in the City edition of the 'Bombay Chronicle' and of the 'Times of India' dated the 17th June, 1941, which together give a fairly full report of what he spoke:

Col. Candy said that as the head of the Indian Medical Service in the Province he was sorry to find that there was not sufficient response from medical men in Bombay to join the I.M.S.

If they did not find the jobs lucrative at least they should have been induced to offer their services from the point of view of humanitarian motives. Besides the war provided a vast scope for gaining both medical and surgical experience.

The Indian Army which was being further expanded, added Col. Candy, required more medical officers to cope with its growing needs. Every Army in the world, be it of any nation, had its own medical corps. The Indian Army too needed Indian doctors to fill its medical corps.

In conclusion, Col. Candy exhorted Bombay medicos to join up in large numbers.—*Bombay Chronicle.*

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An appeal to the members of the medical profession in Bombay to join the Indian Medical Service from a humanitarian point of view was made by Major General R. H. Candy, Surgeon General to the Govt. of Bombay, speaking at a meeting held under the auspices of the British Medical Association, Bombay Branch, at the Bai Motlibai Hospital Lecture Theatre on Monday.

He said that Bombay, which was making a splendid contribution to the war effort, was behind the other provinces in the voluntary recruitment to the Indian Medical Service. With the expansion of the Indian Army a larger number of medical men would be required for service. It was the duty of medical men to look after the Indian soldiers who were fighting so gallantly in the various theatres of war.

Referring to the lack of response in Bombay he said that it might be due to the terms and conditions of service not being sufficiently attractive. But he assured them that there were far greater attractions in the service than mere emoluments.

—*The Times of India.*

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Dr. Jivraj N. Mehta, M.D., M.R.C.P. replied as follows:—

It is good of the Surgeon-General with the Government of Bombay to come over here and address us on the emergency commissions in the Indian Medical Service. He has told us what he wants. Now that he is in our midst and is prepared to meet us and also solve our difficulties, let us be also frank in saying what we may have to say in this connection and speak as man to man.

What is the cause of this hanging back, as the Surgeon-General calls it in his circular letter? On the proper elucidation and the remedying of the factors concerned would depend the success of his campaign. We have always urged and the experience of the last World War and of the present war has proved to the hilt, and to-day's meeting and the Surgeon-General's anxiety are a proof positive of the fact that the only dependable war reserve for medical purposes for the Indian Army is the civil medical profession of India, as it is in England and all over the World and that the War Reserve of the Indian Medical Service as organised on a permanent basis and the Indian Army Reserve Officers in the I.M.S., have proved inadequate and ineffective.

In so far that a substantial element of this service was foisted on the civil medical administrations of the various provinces and with the Central Government as a war reserve, it should justify itself forthwith as a genuine war reserve by reverting to military service. In effecting this reversion to military service, no distinction should be made between the residuary posts in the I.M.S. which are of only recent creation, and the ordinary war reserve. All officers of the Indian Medical Service must give proof of their primary affiliation being to the Army, particularly in a war crisis, and the administration must seek substitutes for them in the Civil Medical profession.

But what do we find? Officers of the Indian Medical Service still continue to be on the civil side. Some of them, though quite young are still continued in civil medical administration,—some of them are still on the staff of the Grant Medical College and in the Medical Schools at Poona and Ahmedabad though it should be perfectly easy to enable the professional and administrative work of these institutions being carried out by the non-I.M.S. element on their respective staff as is done at the Seth G.S. Medical College or at the National Medical College in Bombay.

In the last war Indian medical men, some of them with lucrative practice, rushed to enrol themselves but what a disillusionment it was to them soon as their services became unnecessary on the termination of the war. But it is quite different at the present moment. I am not going to discuss here to what extent the general political situation in the country may be responsible for this lack of enthusiasm amongst the medical men which has brought the Surgeon-General to address us. It has no doubt its share, but there are elements in the country who do not take their bidding, cent per cent, from the political bosses. Even such elements are holding themselves back. Take the Parsis, for example. Though an overwhelming majority of them are true nationalists at heart, they do not follow the Congress, but they, too, are hanging back, only a few having enlisted so far. In the last war they were some of the first recruits to come out and they rushed to enlist themselves in the Indian Medical Service in large numbers.

The fault lies, Mr. Surgeon-General, entirely at the door of the High Command of the Indian Medical Service which treated the Indian element in the service, so soon as there was no use of the Indians for war purpose, in such a downright way that one would think twice over before enrolling oneself in that Service unless circumstances compel one to do so. As the proverb goes, you reap, as you sow. Since the close of the last war, every kind of indignity has been heaped upon the Indian, desirous of being recruited to that Service.

A European could be recruited to it on a permanent basis right from the start, if he so desired. An Indian is offered on the other hand, a short term commission for 5 years, at the end of which, if he has been a good boy, he would be eligible to be considered for a permanent commission. He thus remains under a constant fear of his service being terminated unless he is sufficiently subservient throughout the period of his short term commission. Then the posts of senior and junior specialists in the Service have been created on the civil side and are reserved for the British Officers even though some of the officers so selected may not have had previous real specialist experience and even though quite a large number of Indian officers hold post-graduate medical qualifications, and have sufficient professional experience to serve as Specialists.

The Indian Officer is also being discriminated against by the huge reduction in the number of their recruitment. Prior to the arrangements brought into effect under the Government of India, Defence Department, Resolution No. 205 of 25th March 1937, there were about 386 British Officers in the I.M.S. out of a total strength of about 649; about 263 being Indians. Under the new dispensation the number of British Officers remain the same i.e. 386 out of the total strength of 584 officers, while the Indian Officers have been reduced from 263 to 198. On the civil side, out of 220 officers under the 1937 scheme, there are to be 166 British and only 54 Indian Officers.

Look at the Indian Army list for 1939. There were as many as 455 British Officers in that Service that year as against 386 prior to 1937. So that in spite of the Govt. of India, Defence Department resolution fixing the maximum strength of the British personnel at 386, their strength became 455 in January 1939. Out of the 200 I.M.S. Officers who were given permanent commissions in the I.M.S. in the ten years immediately preceding October 1938, only 33 were Indians.

Out of the 100 permanent commissions given during the period of four years preceding October 1938, less than half a dizen are Indians; very likely three only which means that for years together before the 1937 orders were passed, Indians had practically ceased to be recruited to the I.M.S. with permanent commissions. Between October 1938 and January 1939, as many as 27 British I.M.S. Officers were granted a permanent commission in that service as against a zero Indian during those months.

And you, Sir, harangue us saying that you feel it incredible that young Indians should hand back from military medical service in this war. I may say, that your circular letter in which you describe such an attitude as incredible, is really insulting to young Indians. It is the plans and acts of your own High Command in the Medical Service that are responsible for the meagre response you are at present having in recruitment in this Province. The Indian medical men feel, what's the use of joining a Service where one is going to be treated as the Pariahs of the Service? All lucrative posts, posts to salubrious stations are generally reserved for the Britishers. The station with unpleasant climate or places where there is not much chance of private practice or the Jail Department generally fall to the lot of the Indian Officers.

Look at the Administrative posts? Within about a year or so, there have been, I believe, about 7 changes in the holders of the posts of Surgeon-General and Inspector-General of Civil Hospitals in the different Provinces. Not a single competent Indian can be found to hold any of these posts. When the time for selection approaches, the Indian is ruled out. Oh! he has not sufficient reserve of strength as in the case of Col. Chopra, that distinguished scientist with international fame, though he still continues to be an energetic Head of the School of Tropical Medicine, Calcutta, and of the Biological Standardisation Laboratory. All the vacancies have been filled in by British Officers.

I really regard it as incredible that the British High Command of the Indian Medical Service, after such a black record in their dealings with their Indian colleagues, should expect Indians to come forward to join such a Service. If you want them to come forward to help you in matters medical in this war, let us start on a clean slate.

1. Call up all those I.M.S. Officers at present on the civil side to join the colours straight off.

2. Scrap the civil side of the I.M.S. altogether.

3. Let the Service be a purely military service and the recruitment to it restricted to Indian personnel only. If there are Europeans in India who may want to join it, let them come on the same terms and conditions as Indians and with no privileges. There should be no special or residuary posts for the European officers, and no special terms of employment.

4. The civil medical administration should be under the entire control of the Provincial Government and there should be no correspondence behind the back of Ministers, where Ministries still exist, between the Head of the Civil Medical Department of the Province and the Medical Advisers to the Central Government.

5. As regards the recruits, the terms of employment and the conditions of service, the seniority in Service, the recruitment to the permanent commissions, the gratuity etc. all these should be on the same basis as are applicable to the R.A.M.C. Officers joining the British Army, the scale of pay being according to Indian conditions. It should be also made clear that if any European combatant officers are taken in the Indian Army Units, they must be made to understand that they will receive medical aid at Indian hands.

6. The men volunteering should be started at a rank and seniority in consonance with their experience and standing in the medical profession. All commands should be open to them on this basis of seniority.

7. The scale of pay and promotion must apply to them.

8. All men who take up temporary commissions must have an inalienable right to permanent commissions in the I.M.S. if they wish to go on serving in the army. About half the posts in the provincial civil medical services should be filled by these officers, men being selected on the basis of the excellence of their service record.

9. All officers must be allowed to accumulate their leave and should be given this leave in full on demobilisation.

10. The leaders of the independent medical profession should be on the Board which may deal with recruitment and demobilisation on the termination of the war and they should be closely associated in the selection of officers for the permanent commission.

11. It should be also agreed that the recruitment to the permanent commissions after the war should be restricted entirely to officers holding temporary commission in the I.M.S. and no demobilised R.A.M.C. Officer or a Colonial Officer should be taken in that Service.

We want solemn guarantees for the due fulfilment of these and similar conditions and also for the eschewing of all racial discrimination against Indians.

Sir, we have been asked to be enrolled in the I.M.S. in the spirit of humanity. We have had a very senior officer of that Service addressing us this afternoon and if Dame Rumour is correct, it may not be long before he occupies the coveted post of the D.G., I.M.S. either temporarily or permanently. But let us see what has been the spirit of that service in its relationship with Indians. I shall quote you from what the British Officers of that Service said in their representation to the Islington Commission; a representation which was signed by 20 British Officers on the Bombay cadre, some of them being even on the Professorial staff of the Grant Medical College at the time:—

> "Whilst we recognise that certain appointments should be given to members of the profession belonging to this country when men of recognised ability are available, we would regard with apprehension any "policy of Government which would ultimately jeopardise the appointments at present held by the Service, and which constitute some of its chief attractions. We submit that, in deciding what appointments should be allocated to members of the indigenous profession, care should be exercised that they should in no way be those which might ultimately be regarded as a stepping stone in the higher appointments."

That spirit still pervades in that Service. It was that spirit which was responsible for the horrible treatment meted out to Indian I.MS.. Officers after the termination of the last war. Out of over 1200 temporary Indian Officers who had then volunteered over a hundred only got regular commissions. The remaining who were unceremoniously thrown out, had to restart their lives at an age when they felt inadept for settling down. Among these were officers who were able to come under the eye of the higher commands and were lucky enough to be mentioned in despatches; officers who had received medals, officers who had served for 8, 9, 10 or even 11 years of the best part of their lives—senior enough to be given Major's rank, and officers who were promised a Selection Board for 4 to 5 years, being approved as being good enough for a permanent commission. When the Board was held, these officers after long spells of approved service, were put before the so-called Selection Board and most unceremoniously asked to demobilise and go home, being not selected, while freshers from the Universities and from general practice were preferred. Even the leave they may have accumulated was not allowed to be enjoyed on demobilisation. The fear was that if these tried and trained men in war and peace were retained, they could have claimed seniority to white officers, junior to them, who were either being drafted from the R.A.M.C. or were taken up direct from the Royal Colleges or the Universities. Some men, who had even earned military cross, a distinction which can be won only on the battle field, were rejected. Then, those with many years of loyal service had to face world at a later part of life, when the succeeding generations were being taken up in all civil and military vacancies, and when they found their field of practice choked by the juniors. The common experience of some of these people when they applied for civil appointments was, that the European Head of the Civil Department, including the Railway medical services, adopted a change in attitude which was hardly in consonance to what these people were used to meet with from the same people when the war was on.

It was the generous response of the Indian medical men to take up military service during the last emergency that led to the introduction of racial discrimination in the recruitment to that service. There had never been any racial discrimination, at least not a statutory one, against Indians as regards their entry into the I.M.S. Because Indians responded generously in that war, racial discrimination came into vogue and two Europeans to one Indian ratio was introduced for recruitment and even though before the war the Indian competitors had been getting into the Service by open competition in an increasing proportion, examination after examination, and about the same number of Indians had begun to get into the Service as the Europeans shortly before the 1914 war broke out. Had there been no war, they should have shown a ratio of success higher than even 50 per cent in an open competitive examination held in London.

The 1937 arrangements of recruitment to the I.M.S. smacks of thinly veiled efforts of creating cushy jobs for the Europeans, even at the evident sacrifice of military efficiency. These arrangements also paralyse the civil medical administration. What can you expect when one had in Bombay about 10 Surgeon-Generals (including those in acting leave vacancies) during the course of the last 15 years; the medical policy of the Province not unoften changing with the whims of each new incumbent of the post.

This is the position and if the I.M.S. Officers who desire proper recruitment to their Service are agreeable to see that the very reasonable conditions of recruitment—I have mentioned —are accepted by the Powers that be, I have no doubts that they would be getting a full and free response from a large section of the medical profession in the country.

NOTICE

The attention of the members of the Association is respectfully drawn to the notice on advertisement page v of this issue of the journal regarding the procedure to be adopted in case of non-receipt by them of any issue of the journal.

A. K. SEN,
Secretary, J. I. M. A.

NOTES AND NEWS

ALL-INDIA OBSTETRIC & GYNAECOLOGICAL CONGRESS

The 3rd All-India Obstetric & Gynæcological Congress was held in Calcutta during the Easter Holidays from 10th to 14th April, 1941.

The Obstetric Congress is held under the auspices of one of the four Societies in India one in each of the following places, Bengal, Bombay, Madras including Southern India and Northern India. It was decided to take up the problem of maternity and child welfare in India as the main subject of discussion at Calcutta.

The publicity department of the Calcutta Corporation held a Maternity and Child Welfare Exhibition with the Congress and added immensely to the importance of the problem of maternity and child welfare. Sir Mangaldas V. Mehta of Bombay who is a pioneer in this field and has done much for the welfare of mothers and children in the Presidency of Bombay opened the Exhibition.

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Dr. B. D. Mukherjee, Chairman of the Reception Committee in welcoming the Delegates said:

"Assembled as we are in Calcutta you will naturally want to know from me what we in this part of India are doing for the amelioration of the condition of women and children of the soil. You will have, no doubt, ample opportunities during your stay amidst us to acquaint yourselves with the works of the various maternity and child-welfare institutions in this city. It will not, however, be out of place for me to make a brief mention here of a few of the leading Calcutta institutions which have been doing immense service to the suffering motherhood of Bengal.

Dr. Mukherjee then dwelt on the development of various maternity hospitals in Calcutta and mentioned the name of the following hospitals: Sir Kedarnath Maternity Hospital, Chittaranjan Seva Sadan, Ramkrishna Mission Sishu Mangal Pratisthan, Eden Hospital, Dufferin Hospital and the Corporation Maternity Hospitals.

Continuing, Dr. Mukherjee said, "It will not be out of place, here, I hope, to make a passing reference to the peculiarities of obstetrics in Bengal. It is a notorious fact that women of Bengal lack hopelessly in health, hæmoglobin and musculature. And, the causes are not difficult to find. The climate, the deficient dietary, the appalling poverty of the masses, unemployment amongst the middle classes, ignorance of the rules of health and well-being, social customs leading to sedentarism, intercurrent diseases like malaria, amœbiasis, helmenthiasis and tuberculosis have all contributed to their asthenic condition. It is needless to elaborate here, the effect of asthenia and these intercurrent diseases on pregnancy, labour and puerperium but the net result is the appalling maternal morbidity and mortality which stares the helpless obstetrician glaringly in the face.

"As you are well aware, there are a number of problems which you as obstetricians and gynæcologists are called upon to tackle. I shall, however, conclude with a passing reference to, what in my mind, are the two most important of the many problems which await your consideration. First, and foremost is the problem of removing the colossal ignorance of the mothers and, second is that of providing more and yet more ante-natal and post-natal care to mothers in India."

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Sir P. C. Ray in his inaugural address said,

"Healthy mothers and healthy children are the foundation upon which the entire edifice of a nation's prosperity is built up. It is therefore in the very fitness of things that proper and adequate measures be devised so that the mothers may live well and nurture their children to their full allotted span of life.

"Unfortunately in our country very little attention is paid to the health of women with the result that most of the mothers are very sickly, and after giving birth to two or three children become almost invalidated. It has been estimated that approximately two million women die every year in this country as a result of pregnancy and labour. As nearly ten million live-births take place in British India every year, it is evident that the maternal mortality rate is about 20 per thousand live-births. Whereas the corresponding figure for England and Wales is about 5 deaths per thousand live-births.

"In this modern world, all civilized countries are adopting adequate measures, either through agencies set up by the State or through voluntary organisations formed by members of the public, to secure the physical well-being of every man and woman of the country. Proper ante-natal and post-natal measures are adopted in almost every civilized country in order to protect the mothers from death and disablement and to ensure the birth of healthy and long-lived children.

"In our country the provisions for maternity services are absolutely inadequate and, except in some big cities, very little has been done for expectant mothers. The number of midwives are very few and the number of maternity beds are fewer still. In 1936 in Bengal, there were 84,855 births in Urban areas against which there were only 600 beds. In the rural areas the number of maternity beds were only 33 against 55,84,351 births. In this respect the position in the Punjab, Bombay and Madras was much better. During the same year, there were 633 midwives in Bengal. While in the Punjab there were 2,263; United Province 852; Bombay 2,635 and Madras 1,313. It is quite clear that the position in Bengal is far more deplorable than that in other Provinces.

"It is high time that the Government of Bengal came forward and took up adequate measures for tackling with the grave problem of Maternity and Child-Welfare. We need more medical men and women in this country and many more of them should be persuaded to go to the villages. The Government must take up a systematic programme for training midwives and 'dais' especially for working in the rural areas."

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Dr. N. A. Purandare in the Presidential Address said: "I contend with confidence that our present session in Calcutta will prove even superior to others in view of the energy,

enthusiasm, unity, steadfastness of purpose, eagerness and earnestness in achieving ends, and above all wide-spread fame of the people of this Province for learning and for original and research work. This Province has produced several great men, whose names would ever be remembered with great reverence all over the country. They acted and have been acting as guides and philosophers in almost every walk of life and in varied fields of work, such as political, social, religious, educational and in every profession, not excepting medical."

He paid a glorious tribute to the memory of the late Sir Kedarnath Das whose name was ever uttered in every house in this Province with a deep feeling of reverence and affection, whose skill and ingenuity was so vividly expressed in that glorious instrument devised to requirements for delivering safely the Indian girls in distress from violent pangs of labour, to wit the midwifery forceps named after him, which, he said, would ever remain as a self-raised monument.

He said, "We need more Dases to make rapid marches and effect substantial progress in the province of obstetrics so as to set this ancient country to the forefront in the race obtaining all over the civilized world."

Continuing Dr. Purandare said, "Now that the medical council for India has been brought into being, apprehending how, from want of effective voice of obstetricians, the training of this subject (midwifery) has suffered elsewhere at the hands of their other colleagues, who always form the overwhelming majority and who are ever ready to monopolize a greater part of time of the curriculum for their own subjects, the representatives of the subjects of midwifery and gynæcology on this council from different parts of the country, have out of necessity and duty to co-operate, and working in harmony, to watch closely their own interests. Even here if we now refer to the recommendations of the medical council for India, we will at once find that from the periods of the hospital course nine months have been reserved for medicine and nine months for surgery, while for midwifery, which is recognized as one of the three fundamental subjects, only three months have been most reluctantly set aside; whereas the number of cases to be conducted has been fixed at six. In fact what had happened in Great Britain is being repeated here.

"In these days much has been said about rural uplift and many schemes have been framed or suggested daily by social workers to extend amenities to villagers. No scheme can be complete but includes the maternity service. No work can be perfect but encourages supervision with solicitude of the pregnant woman during her antenatal, natal and postnatal periods. Appreciating that our land consists essentially of villages, our present democratic governments have directed their attention to finding out means to afford medical help to villagers, who have in general no letters, who have never known to raise their voice against any grievance, who bear up quietly and submissively every calamity or malady merely regarding it as a result of wrath of one diety or the other in whose propitiation only lies the remedy. Amidst this profound ignorance no wonder these innocent villagers should have not the least conception about the need of proper care of a lying-in woman. Nevertheless it is incumbent on the popular government as well as on the benevolent social workers to remove this urgent need by finding for them the service of well experinced midwives and medical men. A scheme may be worked out thus.

"Now most of the district towns have each a civil hospital. It should be made the base of the individual district. Some of these hospitals have been expanded with the munificent contributions of the philanthropic rich while the others are likely to be extended in the near future. Thanks to the changed policy of the government, members with high qualifications from the independent medical profession have been appointed to work in them, thus throwing open a scope for honorary workers, thereby indirectly improving the general tone of the profession. Now time has arrived when each Taluka town should have a general hospital with a maternity ward attached to it. Qualified medical men can be found in these days ready to accept honorary posts even in such a hospital. For being in charge of the maternity department one who has secured special qualifications in midwifery and has gained in addition practical training in obstetrics should be chosen. Taking taluka town as the centre, the whole Taluka may be divided into two divisions, one northern and the other southern. In each division may be two medical units, one for the western half and the other for the eastern. Each unit should consist of a medical man, a midwife, a compounder and a boy. Each unit would be responsible for medical relief in all villages comprised in that area. These villages may be distinguished into six groups, the medical unit attending one group in the morning and the other in the evening, so that each group of villages may have medical help twice in a week. To facilitate their going from one place to another, the doctor and midwife may be provided with a small motor car while the compounder and the boy have a cycle each. In each group may be chosen a central place where villagers from the villages of that group may conveniently come for medical advice or to call the doctor or the midwife or both for visiting a patient at home or attending on a lying-in-woman. To carry on the village work, a medical man of lower qualification may be appointed. Their duty, would be to deal with the ordinary run of cases. They should, however, be able to diagnose grave diseases, so that they may encourage the patients to have hospital treatment and may even arrange for their removal, when necessary. The midwife should attend the parturient woman at home in her cottage and might take help of the local dai, who would by observation learn how cleanliness must be observed in the management of normal labour. If the midwife deems necessary, she might send for the medical man working with her to have his advice on certain doubtful matter or to do some such work as applying stitches. She must keep notes of pregnant women in the villages in her charge and call on them in the antenatal period first every fortnight and then every week as term is drawing to its end. Both the doctor and the midwife must submit the report every week of the work they have done to the medical officer in charge of the Taluka hospital. In case of necessity they may seek the help of the honorary obstetrician of the hospital to pay a visit to a pregnant woman in a village; and it would be incumbent on him to do so. At present missionaries are doing yeoman service by rendering medical relief at different centres of each province and the people are deriving immense benefit from it. It is really astonishing when we our-

selves do not volunteer to take upon ourselves this bounden duty which we owe to our own fellow-breathren in our own country and allow foreigners to do this human work for us. Nothing can be more humiliating and nothing does betray so conspicuouly the sheer want of fellow-feeling in us. Moreover, such work is not without any advantage to ourselves; it cultivates knowledge; creates insensibly confidence in us; perfects our skill; at the same time gives us satisfaction of having discharged our duty towards our own countrymen.

"Post-graduate Maternity and Gynæcology Course. In so far as post-graduate teaching institutions are, barring few exceptions, not set up in this country, post-graduates and young practitioners who are anxious to improve their prospects and for that purpose to acquire higher and more practical training, have to visit foreign lands; most of them find it difficult to get enough money to defray the expenses to satisfy their earnest desires. Those who fail to secure pecuniary help cannot carry out their designs and have to rest contented with their present lot. Under these circumstances the establishment of such schools will go a great way in meeting the demand and removing the disability. As has been emphasized before that the need of practical training in obstetrics is the greater in view of the fact that an obstetrician himself has to cope with emergency promptly and adequately. To be qualified for this work a post-graduate institution is the proper place to an obstetrician just as a workshop to a workman to be a skilful craftsman. To man such an institution whether meant for the study of medicine, surgery or obstetrics, now no difficulty should be encountered when men of eminence, ripe experience both in teaching and in executing work practically are available all over the country."

GOVERNMENT MEDICAL SUPPLIES

In connection with reports about the Government of India placing orders for three years' stock of medical supplies from England on behalf of certain Provincial Governments, the Indian Chemical Manufacturers' Association has addressed a communication to the Government urging the Government to ascertain from the Indian manufacturers about items that they can supply at present or in near future before orders in England are placed. The Association regrets that although the suggestion for ordering three years' supplies to Provincial Governments was made by the Government of India, sometime back the indigenous industry was not consulted as to the items that it can supply. It is pointed out that India produces almost all the standard medicines conforming to the B.P. and several patent medicines, etc. The proposed step of the Government of India in placing long term order would, therefore, prove detrimental to the Industry and would also put a stop to new ventures. The Association further points out that in placing orders in India for medical supplies the Government of India have been giving short period orders. It is suggested that instead of placing long period orders in England the Government should help the progress of the indigenous industry by placing at its disposal an assured market for a long period.

The Association has also addressed letters to all the Provincial Governments enquiring as to whether they have agreed to the suggestion of the Government of India in this matter and in that case also requesting the Governments to enquire from the Association as to whether the items for which orders are proposed to be placed are available in this country.

MEDICAL RELIEF IN CERTAIN RIOT-AFFECTED AREAS IN THE DISTRICT OF DACCA

On a request from the people of certain riot-affected areas in the district of Dacca, Dr. K. S. Ray, the President of the Indian Medical Association, deputed Dr. A. K. Chakrabarty, M.B., Hon. Jt. Secretary (Headquarters) of the I.M.A., to visit these places and render immediate aid to the victims.

Dr. Chakrabarty accompanied by Dr. Biren Bose of the National Medical Institute reached Dacca on the 2nd May, 1941 and met Mr. Blair the Commissioner, and Mr. George, the Magistrate, and they were informed that there were no epidemics of cholera, typhoid etc., rampant either in Dacca or suburbs. The information was confirmed on local enquiries and the ampoules of Cholera Prophylactic Vaccine received from the Public Health Department were subsequently returned to the department.

Dr. Chakrabarty next went to Narsingdi, where also on enquiry no epidemics were reported. He was, however, informed that about 26 doctors had lost their instruments and other medical accessories and the stock of stethoscopes, syringes, needles etc., were distributed among them through Dr. Benimadhab Bose of Purana Paltan, Ramna. In all 15 doctors were each supplied with a stethoscope, a 2 c.c. syringe, 3 needles and one thermometer, while nine others were supplied with a 2 c.c. syringe, 3 needles and one thermometer each.

Three 100 c.c. syringes have been kept in the relief centre to be distributed to three qualified doctors later. Three boxes of Mixed Influenza Vaccine, 200 ampoules each of Cholera and Dysentery Phage, Intesti phage and Bi-Cholera phage have also been kept with Mr. Srish Chatterjee at the relief centre.

The President of the I.M.A. wishes to express his gratefulness to Dr. B. C. Roy and Mr. Nalini Ranjan Sarkar for having made available the materials for distribution among the members of the medical profession and the public of the riot-affected areas.

OBITUARY

DR. BHAGWANDAS S. MANGHIRMALANI

Death has occurred of Dr. Bhagwandas S. Manghirmalani, M.B., B.S., D.P.H., D.T.M., an esteemed member of the Karachi Medical Association (Sind Provincial Branch, I.M.A.) on the 15th April, 1941. at the early age of 44.

Dr. Manghirmalani graduated from the Bombay University in 1919 and joined the Municipal Service in 1921 as medical officer in charge of the Epidemic Hospital. In 1926 he obtained his D.P.H. and D.T.M. from an English University. He was later appointed as Deputy Health Officer and very recently as Assistant Health Officer of the Municipal Corporation, Karachi.

His pleasing manners and winning smile endeared him to every one he came in contact with. His death has cast a gloom all over the city which has lost one of the ablest officers of its Health Department.

The Karachi Medical Association passed a resolution mourning his loss on the 18th April, 1941.